Rykerr Medical's

Vent Management Guide

for Invasive Mechanical Ventilation in Transport

Version 2 Draft for Peer Review
Aug 2026

Intro to the Version 2 Draft

Version 1 of this here Vent Book went live in May 2020 and it's high time for some edits and an updated version that (hopefully) will clarify a few things. We also have added some stuff that we didn't directly address in the first version. Another factor is the widespread prevalence of the Hamilton T1 since the publication of the first version, which definitely warrants discussion of a few things we didn't cover before. And we've received lots of feedback to date and we've tried to incorporate it all into the final product.

In an effort to open this process up to everyone in the community we've opted to track all the changes from the final Version 1 to Version 2 in this Google Doc. In addition, this document is open to comment for anyone with the link - all readers should feel free and welcome to add notes in an effort to facilitate this process. All of us will be able to both track changes and view comment history with this format. If the Google Doc thing isn't amenable to anyone's lifestyle and/ or technical prowess, we're still open for feedback by email, via the website, on Instagram or Twitter, and even by way of smoke signals.

To see the progress of this thing from the very beginning (to include hand-written edits on a final copy of Version 1) take a look on our site: Vent Management page, scroll down to "The Process."

Summary of changes:

- lots of wordsmithing that doesn't necessary change the material, but makes it all more readable
- added section titled "vent anatomy" to review parts of the system(s) we use to deliver mechanical ventilation
- included the concept of volutrauma vs. simply barotrauma and injury in a general sense
- expanded discussion of PEEP to include how to choose and titrate values
- expanded on and relocated discussion of $EtCO_2$ as it relates to $PaCO_2$, specifically the gap between these values
- significant revision of the section on comfort
- added mention about $EtCO_2$ waveform and vent graphics into discussion of obstruction strategy
- updated our recommendation on tube clamping to prevent derecruitment (end-exhalation vs end-inspiration)
- clarified recommendations for management of pneumonia - previously had a note about using PEEP specifically, now refining that to simply stick with a lung injury strategy in general
- created section called "initiating ventilation" to discuss the process and initial steps after transferring a patient onto the transport vent
- rewrite of section on acute deterioration
- added section on pediatric ventilation
- created vent graphics section to cover waveforms & loops as well as dyssynchrony patterns
- third additional concepts section to cover expiratory time constant, blood gas analysis, weaning (and pressure support) and APRV
- reviewed many of the references and replaced some with more current literature
- in general, steered more towards concrete recommendations vs. simply presenting concepts neutrally (i.e., more focus on AC > SIMV, an explicit outline for a peds strategy, etc.)
- also adjusted (just a smidge!) recommendations for when/ how often to check things and how to set limits/ alarms
- redid all the math stuff and caught a few errors in calculations (and associated claims) from the first go-round, nothing major though

Let us know what you think about it!

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Legal Statement

Rykerr Medical LLC presents all of this good stuff for education purposes only. While we hope that the content here will be of use to our audience in the clinical setting, all actions taken by consumers of this information is at the discretion of the individual and should be in accordance with organizational policies, professional scope of practice, appropriate medical direction, and/ or current best practice. We have done our best to provide references for all ideas not our own and to utilize sources that are both truthful and reputable.

A Personal Intro

There are a lot of good reasons why I thought it'd be good to put together a primer on vent management, but the main one is that my first vent experience was a near-disaster and I'd like to share what I've learned since then so that others can avoid what I had to go through. I also think there's some room for diversity in how we, as an industry, present material to each other and move forward with our understanding of complicated things. And lastly, I hope that this interactive style of writing can be of help to some folks and maybe inspire others to do the same and build on the whole idea. Collaborating together to improve our skills and holding one another accountable to live that out through clinical practice means better care for more people.

But to start with the awful beginning story: I was brand new to an ambulance service in New Mexico, having moved from Pittsburgh about two years after I first got my medic. I was still green but felt like I had gotten a lot of experience back in the city and was maybe a tad over-confident. Anyway, I started at this rural service in mid-November and the call that prompted my journey down this path of ventilator self-education was the day after Thanksgiving. I had basically just arrived in NM, barely gotten settled into the second EMS service I had ever been given medical control at, and was turned loose to practice in the field.

Things were different for sure. Five- and ten-minute transport times had been replaced by ones that sometimes took hours. With a five thousand square mile coverage area, the ambulances were giant machines that could be rigged to carry three patients each and would never have made it in the city alleys. Protocols and capabilities were a lot more lenient and included vents, surgical crics, hiking to patients broken in the woods - that sort of thing that this city boy just hadn't done before.

Oh, and also two-patient interfacility transfers. Our flagship hospital was in Albuquerque, one hundred and eighty miles or two and a half hours away by bus, so it was hugely advantageous to load two patients in on a single truck to avoid an extra six-ish hours of that second truck being gone from the service area. And when I was asked if I was OK with a vent patient and a psych patient going up to Albuquerque at the same time I didn't turn it down and we started getting things together. Part of that prep process was another guy showing this guy how to use the LTV1200, as I hadn't gotten to that part in my orientation and didn't yet have the confidence to say "no" to things I wasn't comfortable with or ready for.

My five-minute vent lesson was subpar, to say the least, and then I was off to the big city with the vent patient on the stretcher and the psych patient on the bench seat, two EMTs up front just in case I needed anything. My first action when the vent started beeping was to press that handy silence button – per the lesson I had received on the machine's operation. When that didn't work I figured it might be because the patient wasn't listening to the vent settings we had dialed in before leaving, so I paralyzed him – also per the lesson I had received. And that worked for a little while. Then I started getting more alarms and a low sat, so I did what all good medics do and disconnected the vent, grabbed my BVM and had the EMTs up front pull over so that one of them could hop in the back and give me a hand.

Sats stayed low, the alarms were yelling at me, the EMT was like, "come on, bro, get it together," and I didn't know what to do. So I turned the vent off, pulled the tube out, and started over from the very beginning with BLS airways and the BVM. That happened, we had the airway secured, sats came up, and then I handed the bag off to the EMT and set my sights on restarting this vent machine the way I had been taught just a little while ago. It was during this process that I realized my connections from the machine to the circuit had come undone. I must have stepped on them or something during the shuffle… Nowadays I would have simply looked at which alarm I was getting and worked through a systematic process for addressing it. The whole fiasco would have been avoided. But back then I didn't know a single thing about vents, to include that the text on the screen was relevant to getting the alarm to stop.

And that's just part of the story. One other part, don't forget, is that other patient on the bench seat watching the whole damn thing and me hoping he stays cool enough that I don't have to try and manage two patients simultaneously. Another part is that even though I finally did get that alarm situation sorted, I still had trouble managing my vent settings. I couldn't maximize my SpO_2 or keep my $EtCO_2$ in range, my patient would get super agitated every time the Vec wore off, etc.… We did, however and finally, arrive to the big city

in a presentable state – perfusion was good, sats weren't embarrassing, the patient appeared comfortable enough and was making some effort to breathe on his own, and that machine had stopped yelling alarms at me.

With the patient handoff complete, I returned back to small town New Mexico late on that day after Thanksgiving, year 2012, and decided then and there that I was never, ever, going to be in that situation again. My initial study list looked something like this:

The Ventilator Book by William Owens

The LTV1200 Product Manual (and the DVDs)

EMCrit Dominating the Vent Series
Part 1, Part 2

I later came across many other great resources and I will mention those as we get to them. And also, I got on the technology train. Which I think is a huge facilitator of learning when used in the right way and I hope that this little experiment can demonstrate that. In this eBook version, all references are linked via footnotes – just click the little numbers to open the notes, then follow the links from there to see both external sites and internal sections within the document. And if you have a version where the links don't work because it isn't legit, that's cool too: visit us at rykerrmedical.com where you can download a free pdf version.

So now let's jump into the weeds and see where we end up. Keep in mind that this is to be an ongoing project and my first foray into this type of thing – so if you have feedback, just send it my way or offer to lend a hand. I'd love to get more folks involved in this and to make it both better and more accessible for everyone.

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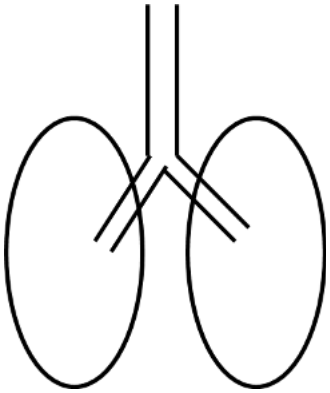
Some Very Basic Physiology

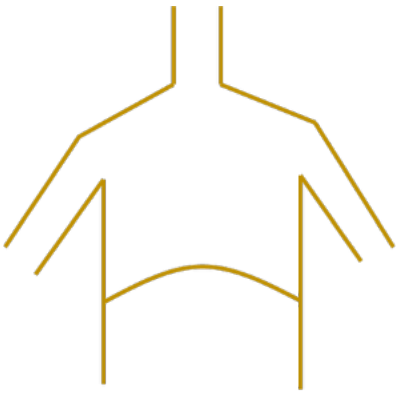
Before discussing mechanical ventilation, we need to cover fundamental concepts of the respiratory system and the breathing process. The stuff outlined here is intended only to give a foundation for understanding vent management. One recommendation for looking into the details beyond this (much of which comes up later when we talk about specific conditions) is a good, solid, heavy Anatomy and Physiology textbook or any of the references noted.[1]

The Normal Breathing Process

Let's start with a picture of the major components involved in normal inhalation and exhalation. At its most basic, we have the lungs and the large airways:

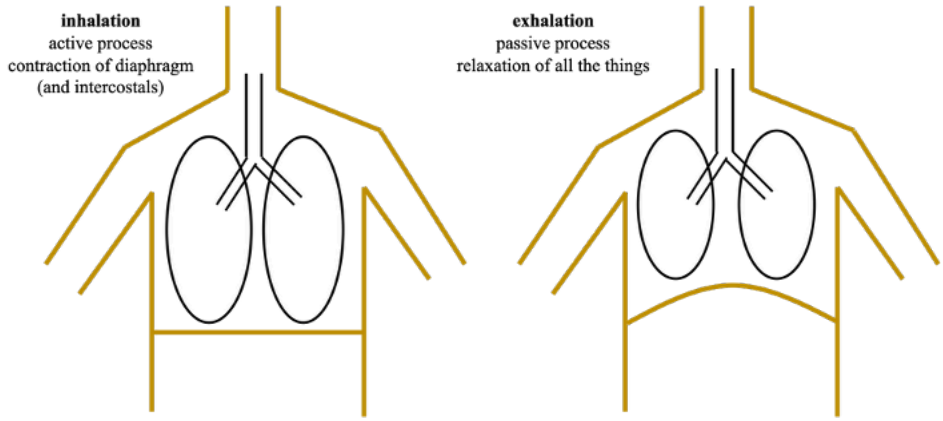


We also have the chest cavity and the diaphragm:

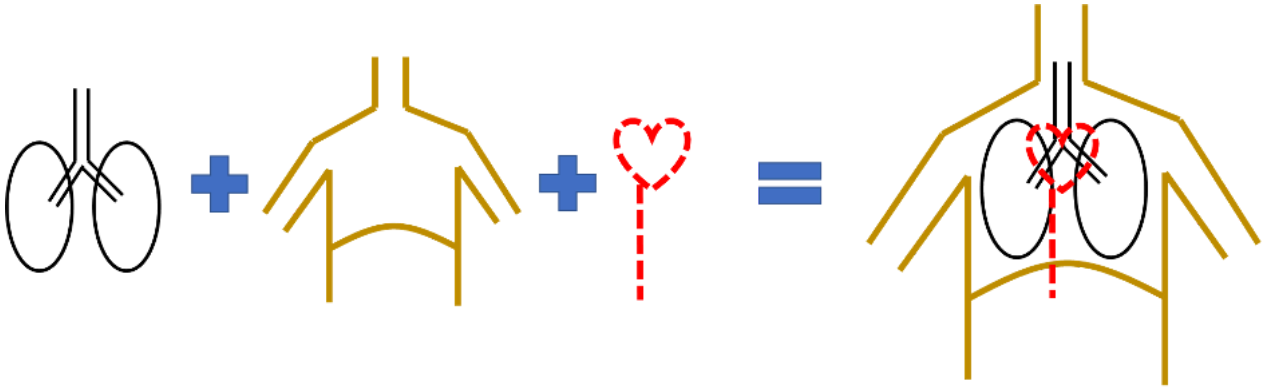


[1] Also see **Suggestions for Further Study** at the end for more content on both vent management and the physiology behind it

The lungs are attached to the chest cavity and diaphragm so that when the diaphragm contracts or flattens, the lungs expand – this sucks air into the lungs via a negative pressure:



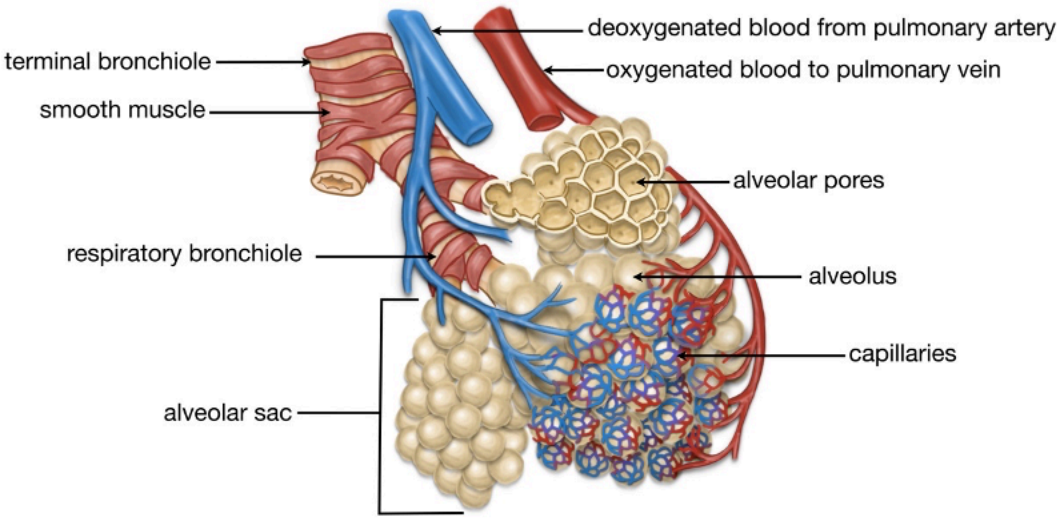
Inside this same cavity lie the heart and great vessels; most important to our discussion is the inferior vena cava:



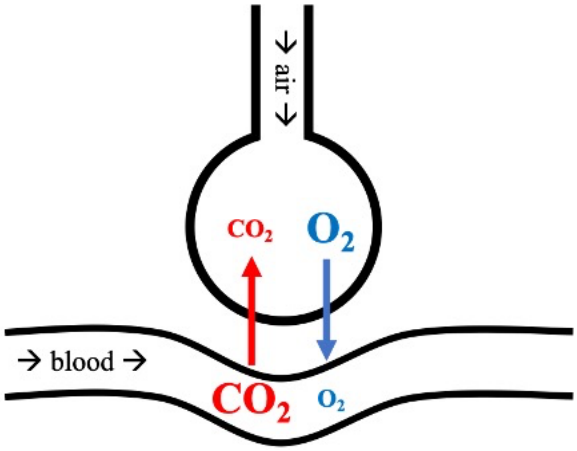
Thus we have a system that normally functions by contraction of the diaphragm (with or without help from the intercostal muscles) to create a negative pressure, sucking of air into the lungs.[2] Because this air movement occurs via negative pressure, blood return via the inferior vena cava is facilitated by normal ventilation.[3] This will be important when we move on to talk about positive-pressure ventilation in just a minute.

[2] Hasudungan, 2014 - To review the physiology of breathing in a bit more detail, refer to this video
[3] Azizov, 2017 – Another video that explains the mechanism by which normal breathing supports cardiovascular processes

Zooming in, the image below shows blood vessels encircling little sacs, known as alveoli, which are the homestay of pulmonary gas exchange where oxygen (O_2) goes into the blood and carbon dioxide (CO_2) comes out:[4]

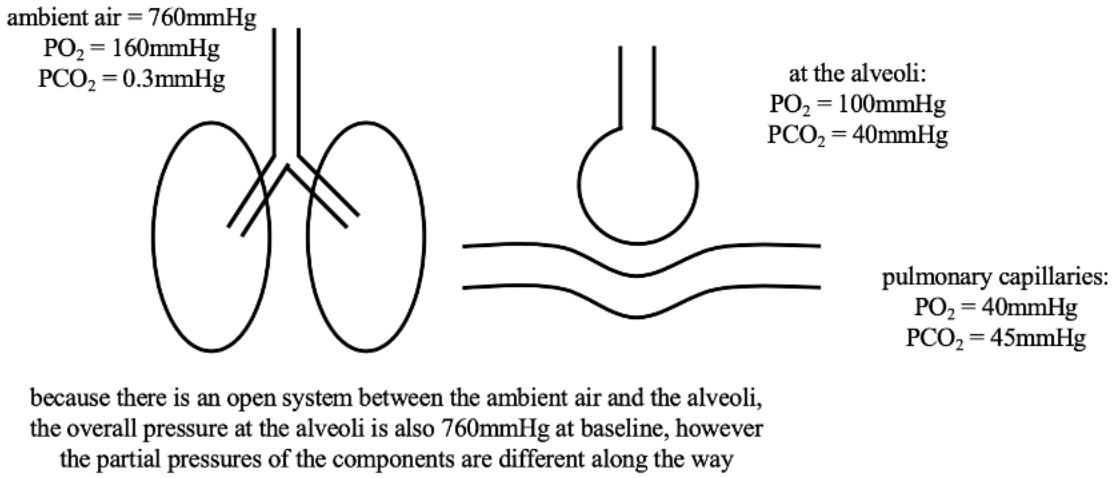


A simplified version of a single alveoli with a corresponding blood supply can help us understand the pathophysiology of different situations:



[4] Graphic built by Dr. Sameer of *Art by Dr. Sam* on Fiverr and *The Young Orthopod* on YouTube

Next, let's add some numbers to that graphic of a single alveoli and its blood supply.[5] Note that in real life blood is continually moving past the alveoli and gases are constantly shifting to reach equilibrium, so that as carbon dioxide is offloaded and oxygen is onloaded, there is a new supply of blood and a reset of the gradients across that membrane. Plus the diffusion of gases from alveoli to pulmonary capillaries happens very quickly. This means we generally aren't worried about this timeframe (i.e., how fast these gases diffuse) being the limiting factor in this process:[6]



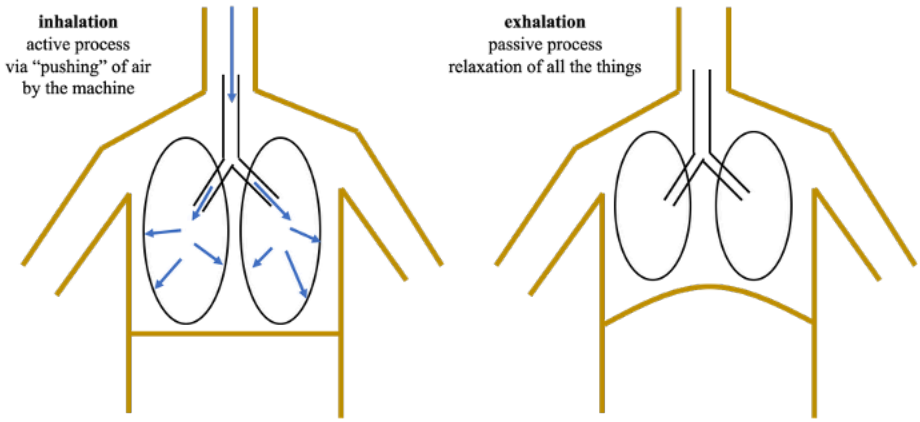
The pressure gradient or difference from alveolus to capillary is drastically different when comparing the two gases: oxygen has a pressure difference of about 60mmHg, carbon dioxide has one of just 5mmHg. While this may seem, at first glance, to put the body at risk of some sort of imbalance, carbon dioxide moves more easily through the liquid membrane (roughly twenty times so) and the net result is that oxygen and carbon dioxide exchange at about the same rate.

[5] Betts & friends, 2013 - This chapter discusses these numbers in the context of Dalton's Law and lists all the values we used except for PO_2 at the alveoli; that one is cited as 104mmHg in their text, but we calculated it out in the **Appendix** and use our calculated value to maintain consistency throughout this text

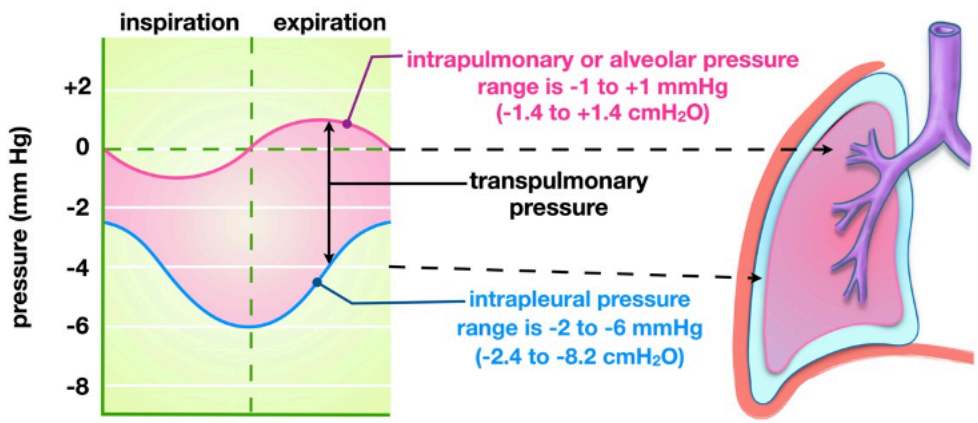
[6] Speller, 2018 - Outlines how both oxygen and carbon dioxide diffuse in the pulmonary system within the context of gas laws; do note, however, that certain states can slow this process down (and we'll get to those later on)

How is Positive-Pressure Ventilation Different?

Now we need to consider what happens when we bypass the whole negative-pressure mechanism for ventilation and instead opt for a positive-pressure approach.[7] Let's start at the top with the basic sketch of airways and lungs superimposed on the chest wall and diaphragm. When we ventilate by positive-pressure ventilation (PPV) we have to physically displace the diaphragm and chest wall while simultaneously pushing air into the system. This requires a lot more pressure than we needed for that negative-pressure, spontaneous mechanism:



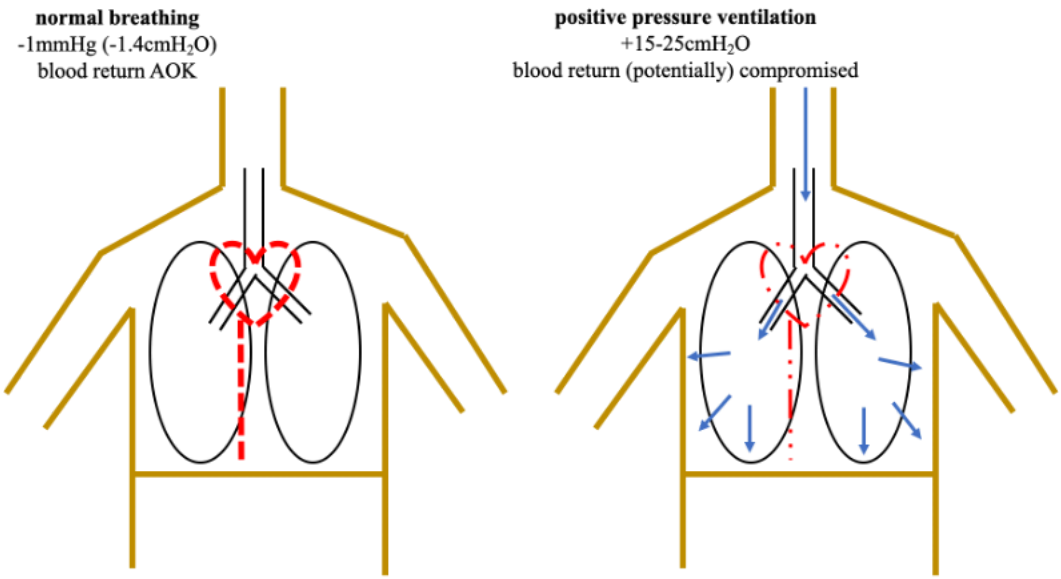
We will get to airway pressures and limits for them later on, but a normal **Plateau Pressure** (which reflects the average alveolar pressure in positive-pressure ventilation) is in the range of 15-25cmH_2O; compare this to the pressures represented in the following illustration:[8]



[7] This assumes that the patient is not contributing to this effort of breathing; to say it another way, this description is accurate for the patient who is not making any respiratory effort or is out of synch with mechanical efforts; in reality we can synch patient effort to machine effort to minimize the differences and effects discussed in this section (more on this in **Comfort** and in **Vent Graphics**)

[8] We'll talk about the mmHg and cmH_2O conundrum at the end of the next section (in **Measuring Pressures**); alveolar pressure is the most relevant to our discussion for now, the concepts of transpulmonary pressure and intrapleural pressures are deferred here; graphic built by Dr. Sameer of *Art by Dr. Sam* on Fiverr and *The Young Orthopod* on YouTube

The biggest impact of that increased intrathoracic pressure is the effect it can have on cardiac output. Increased intrathoracic pressure can decrease blood return to the heart via pressure on the vena cava, resulting in decreased preload and, therefore, less output.[9] Let's represent it this way:



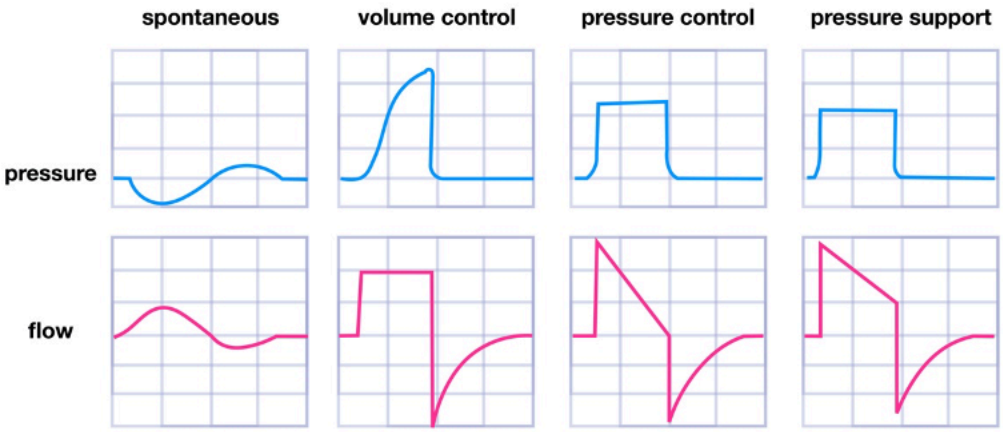
Other negative sequelae of positive-pressure ventilation (which may still occur even if we have all the settings dialed in right!) would be patient discomfort, muscle fatigue and/ or weakening,[10] and physiologic changes to other body systems.[11] And then if we have things dialed in wrong on the machine or don't ventilate appropriately based on patient presentation, we can also cause things like direct injury to the alveoli and hypoventilation (leading to shock). This is but a short list of the major things we'll worry about in this manual, just recognize that there is a lot of potential for bad and that's why we need to know how to manage the machine to the best of our collective ability and to mitigate as many of these things as we can along the way.

[9] Strong, 2013; Mahmood & Pinsky, 2018 - Both this video and the article explain in more detail on how positive-pressure ventilation (and particularly **Positive End-Expiratory Pressure**, discussed later) can affect cardiac output, especially with concurrent hypovolemia; while it isn't always true that positive pressure decreases cardiac output (sometimes the opposite can occur), the positive-pressure ventilation/ PEEP → decreased preload → decreased cardiac output sequence of events is most relevant to us in the transport setting

[10] Tobin & friends, 2010 - Outlines the idea that we can mitigate fatigue and weakening by adjusting vent settings to require that the patient make some intrinsic effort to breathe; while their ending advice is to utilize an airway pressure waveform to monitor patient effort (something we don't always have in the transport setting), it still provides valuable insight on the whole concept; more on that in **Vent Graphics**

[11] Yartsev, 2023b - In fact, navigate to the *Respiratory System* header at the top of this page and then down to the section on *Physiology of Positive Pressure Ventilation* for more detail on all of this stuff

We already saw how a pressure waveform might look over time with spontaneous, negative-pressure breaths, so let's see how it looks with machine-delivered ones. Note that there are different types of machine-delivered breaths in this diagram (plus some terms to discuss), and we haven't yet gotten there; that's totally OK and we just want to point out some general trends here. Big takeaway: the left set of patterns (the normal) looks nice and smooth, without any harsh changes or drastic swings in amplitude; all of the others have those things we don't want. Another noteworthy point is that the graphic representations of the types of breaths (i.e., each column of the three towards the right) are each slightly different. Sometimes one mode or type of breath will be more comfortable for a certain patient in spite of trying to do all the other things we know how to do, simply because how that patient's body responds:[12]



In an effort not to discourage anyone from ever putting a patient on a vent, there are advantages to positive-pressure ventilation and mechanical ventilation. Most obvious of these is that it allows us to breathe for a patient in a relatively simple way when that patient is unable to do so on his or her own. More specifically, mechanical ventilation allows us to control and direct recovery with specific conditions and diseases (such as acidosis, asthma, and ARDS – all of which we will discuss later on). Positive pressure can help move oxygen into the bloodstream more easily, managing ventilation can help that oxygen get delivered to tissues more effectively, manipulating time spent at different parts of the respiratory cycle can increase the amount of time that the body can participate in pulmonary respiration, etc. There are lots of good uses of the ventilator and we will get to all of them in due time, so don't worry if that got to be too much for a moment and know that in spite of its drawbacks, mechanical ventilation does have its place in the cosmos.[13]

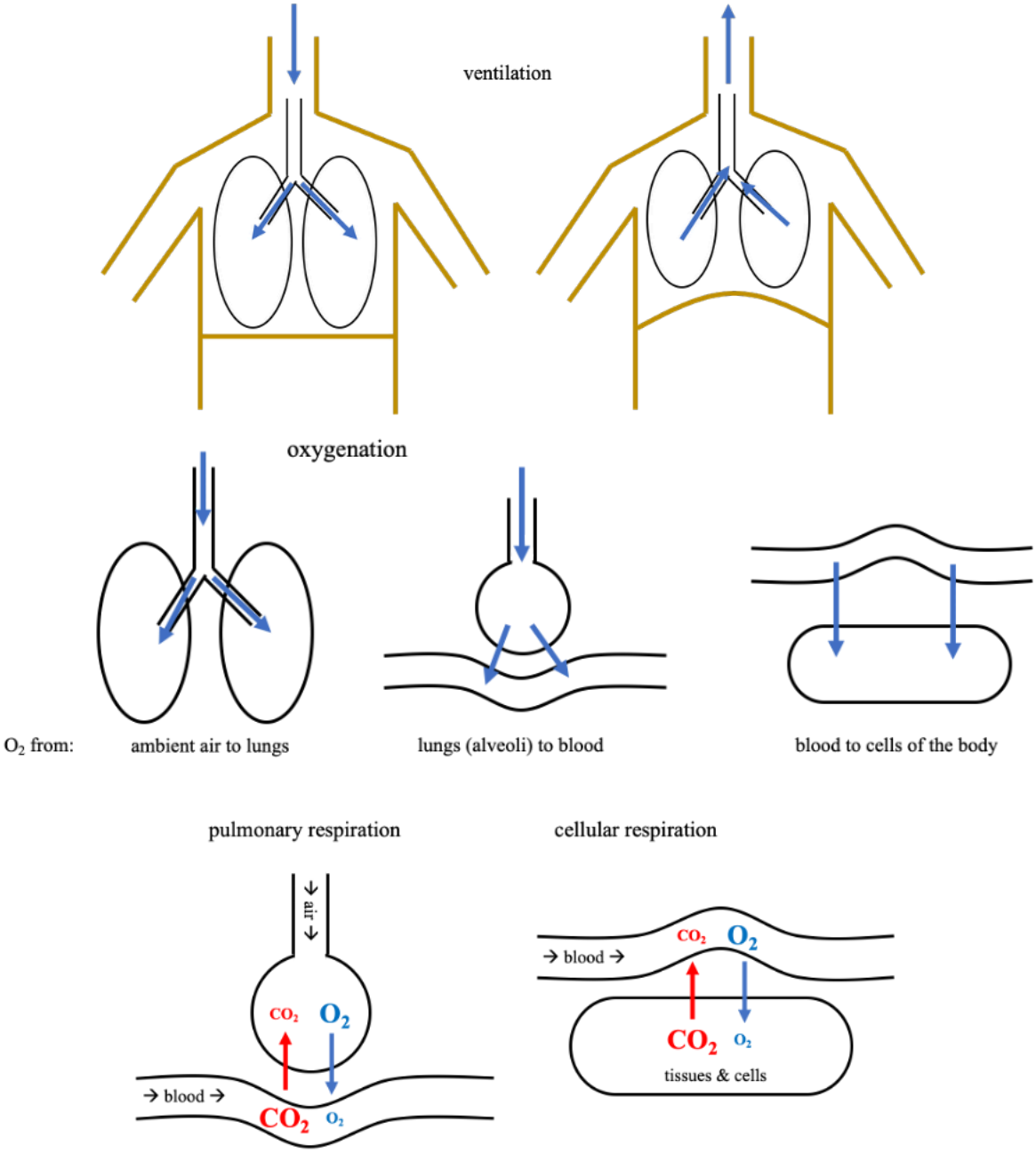
[12]This assessment of what the body wants in terms of smooth waveforms and avoidance of harsh changes in amplitude is a subjective concept - it seems to make intuitive sense, but there may not be a good way to verify the idea; graphic built by Dr. Sameer of *Art by Dr. Sam* on Fiverr and *The Young Orthopod* on YouTube

[13] Hill, 2019 - And if someone needs convincing that mechanical ventilation is preferred to simply using a bag-valve device, take a look at this discussion of a recent paper

Other Important Concepts

Terms to Describe Breathing

There are a number of ways to describe breathing, but they are not all interchangeable.[14] **Ventilation** refers to the gross movement of air as the body breathes in and out. **Oxygenation** refers to the transition of oxygen from the air outside of the body, through the respiratory and circulatory systems, and to the capillaries where it can be picked up by the tissues for use. Lastly is respiration, which has two specific types. Pulmonary respiration refers to the exchange of carbon dioxide and oxygen in the alveoli of the lungs; cellular respiration refers to a comparable gas exchange at the tissues. To visualize it all, here are a few images:



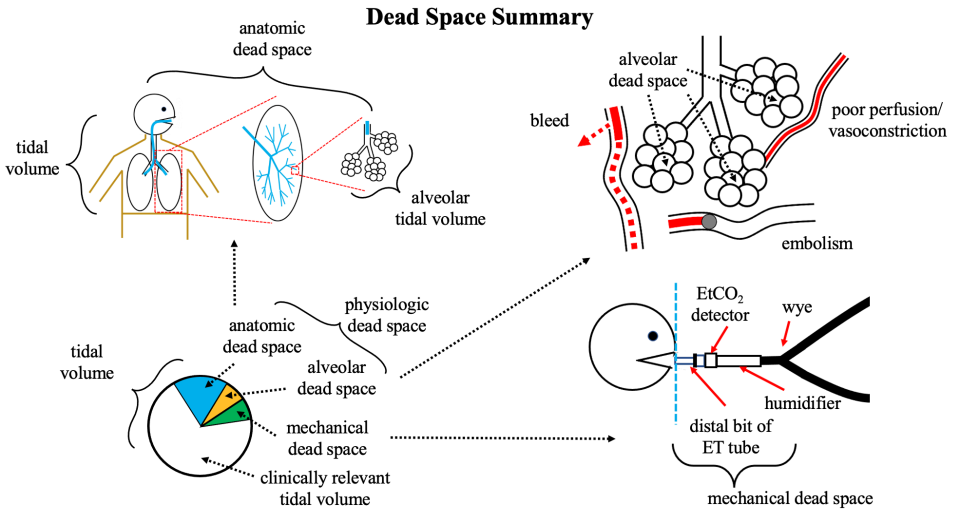
[14] Betts & friends, 2013 – Explains in more detail the processes of ventilation (Section 22.3) and respiration (Section 22.4)

There is some overlap between oxygenation and pulmonary respiration in all of this, but it helps to separate these ideas. When managing the vent, we typically focus on the processes of ventilation and oxygenation. While respiration (in both forms) is very important, our ability to manipulate it isn't as straightforward as it is with ventilation and oxygenation. That said, we can actually influence all of these processes with the ventilator and we will point these things out as we get to them.[15]

Dead Space

Dead space can be an intimidating concept in vent management and we are going to try to both simplify it and identify specific situations in which it matters. To start with, there are four types of dead space that we will discuss: anatomic, alveolar, physiologic, and mechanical.[16] Dead space, as a term, can be used to describe any one of these subtypes, but it helps to recognize which type of dead space is of concern in a given situation. And know that we sometimes see dead space notated as V_D.

Let's start with a summary graphic, then we'll discuss each of the types in detail:[17]

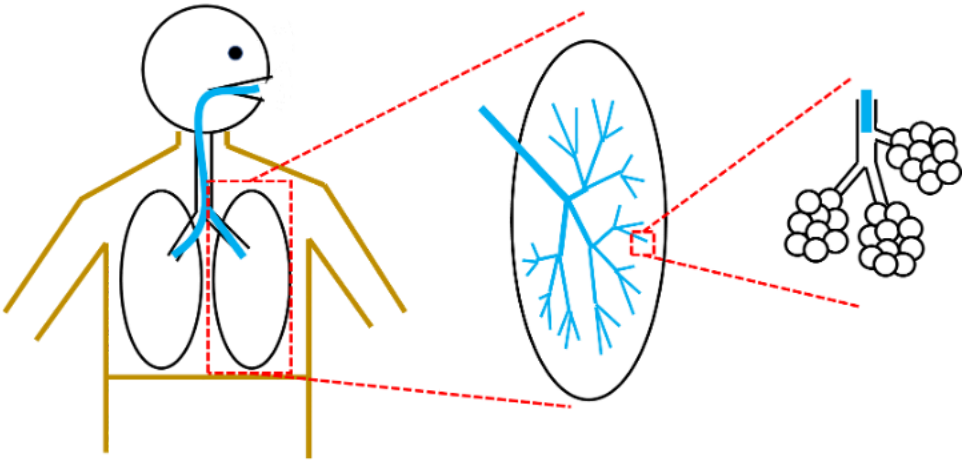


[15] Hasudungan, 2018 - This video is a general recap of respiratory system physiology and the oxygen-hemoglobin dissociation curve, it sheds some insight on this idea that ventilation is intricately linked to both pulmonary and cellular respiration; we will reference it again when we discuss **Oxygenation**

[16] Yartsev, 2023a - This is the best content we've been able to find on this subject of dead space, very thorough and with references to more information along the way

[17] This graphic is from our blog *Dead Space Illustrations*, refer there for more detail on this concept and actions that can be taken to minimize the clinical impact of dead space in its various forms

First of all, anatomic dead space is the air involved in the respiratory cycle that cannot participate in gas exchange. As represented by the blue lines, it starts at the naso- and oro-pharynx and extends down to the terminal bronchioles:



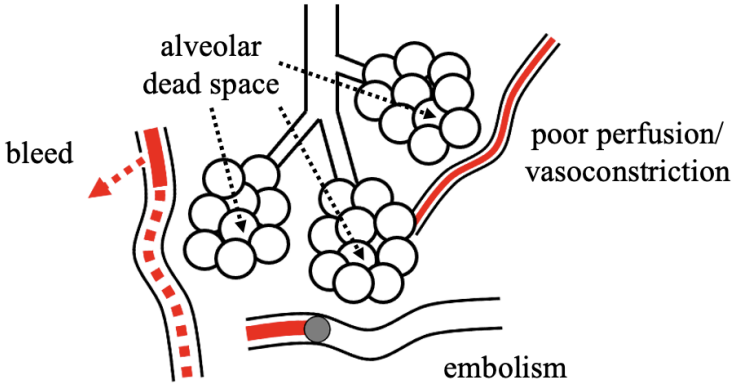
Another way to describe anatomic dead space, in light of this graphic, would be just about all the air involved in a breath other than what ends up in the alveoli and respiratory bronchioles. Now this graphic isn't to scale, so it sort of seems as if dead space is the majority of the air involved, but that isn't the case. There are over a thousand terminal bronchioles in a single lung and hundreds of millions of alveoli total, so the majority of air ends up in the alveoli.[18] It's also worth noting that this process is dynamic and that anatomic dead space refers to the air outside of the alveoli and respiratory bronchioles when those alveoli are fully inflated at peak inspiration.

Anatomic dead space is most relevant in our discussion of ventilated patients when we need to alter the amount of air that participates in alveolar gas exchange (i.e., **Ventilation**). We will talk about this more later, but we basically have two options when it comes to increasing this amount of air: increasing the frequency at which we deliver breaths or increasing the amount of air per breath delivered. If we add one breath to the equation, we must consider anatomic dead space and therefore the amount of air to the alveoli is less than the actual volume of that entire breath. On the other hand, if we simply add volume to breaths already being delivered, we get more of that additional volume at the alveoli because anatomic dead space has already been considered for each breath.[19]

[18] Betts & friends, 2013; Ochs & friends, 2003 - And just to clarify: the terminal bronchioles (marked by the thick blue line in the far right side of this graphic) are different than the respiratory bronchioles, which are the stems distal to that blue line that feed into each cluster of alveoli

[19] There are some nuances to this idea of how dead space varies with volume delivered, but we'll cover those later on in **Volume Control**, **Tidal Volume**, and the **Appendix**

The next type of dead space is alveolar dead space. Alveolar dead space refers to the air in the alveoli without a corresponding blood supply.[20] The classic example is an embolus that restricts blood flow to a portion of the lung. Other causes would include decreased cardiac output (i.e., shock) and chronic lung disease (i.e., COPD).

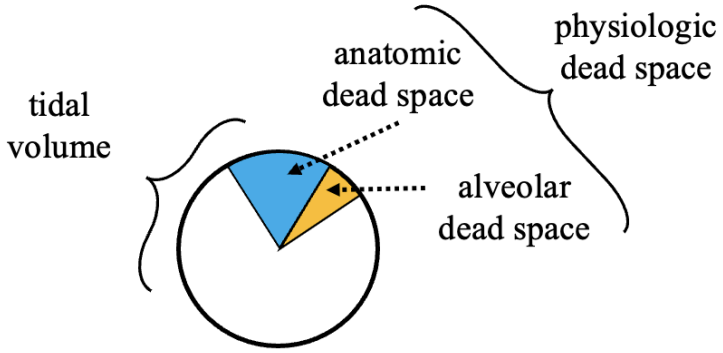


It is worth mentioning that alveolar dead space is often described as an absolute thing, as in zero perfusion to a particular alveolus. In reality, however, there is a spectrum on which ventilation and perfusion can correlate. The details on that and a quantitative definition don't matter much in this discussion, but it is good to know that there is some variation in defining alveolar dead space.

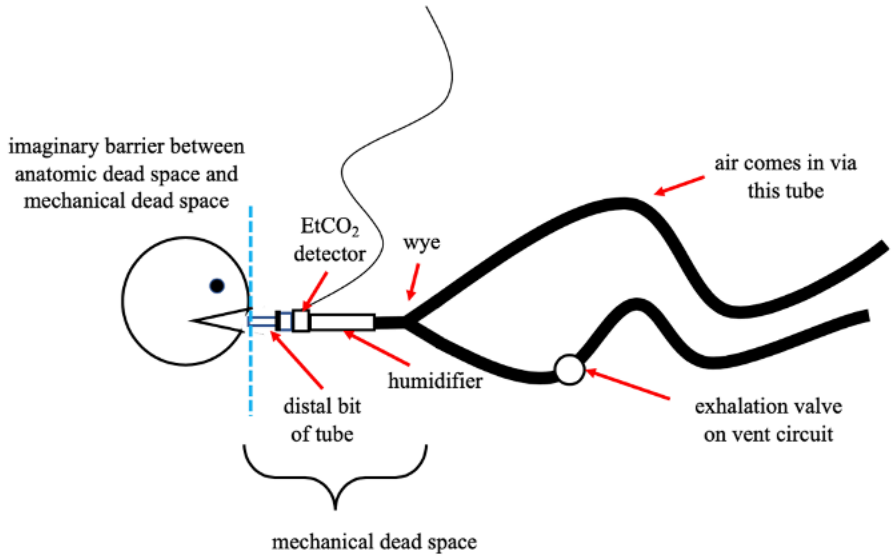
In the normal human body, alveolar dead space is close to zero and we consider it to be negligible. For the sick or injured, however, we assume some alveolar dead space and proactively take steps to accommodate that. These actions, all of which we will touch on as we move forward, include ensuring adequate oxygenation,[21] judiciously applying end-expiratory pressure,[22] positioning the patient properly, and maintaining hemodynamic function to avoid shock states.[23] All of these things collectively maximize the efficacy of the ventilator treatment we are administering. In the event that no alveolar dead space existed before these interventions, no harm is done. All of these things are of benefit even in the absence of alveolar dead space, but they are particularly beneficial for those patients who do have some degree of it.

[20] Mason, 2019 - For an alternative explanation of alveolar dead space, refer to this short video
[21] This idea of mitigating dead space via **Oxygenation** ties into the very next section on **Hypoxic Pulmonary Vasoconstriction**
[22] Murias & friends, 2014 - As outlined in this article, **Positive End-Expiratory Pressure** can reduce alveolar dead space, but only to a certain point; after that, further increases will create more of it
[23] Yartsev, 2023a - Among many other things, this article describes how we can reduce alveolar dead space by both positioning the patient appropriately and maintaining perfusion

Next on the list is physiologic dead space. Physiologic dead space is the sum of anatomic dead space and alveolar dead space and represents all of the dead space before we introduce our devices into the system. In the healthy person, we often assume no alveolar dead space and therefore physiologic dead space is equal to anatomic dead space. The utility of knowing physiologic dead space is that it can be measured and then used to calculate or estimate those other two kinds of dead space: alveolar and anatomic.[24] But given that this isn't normally a possibility in transport, the utility of describing dead space this way doesn't much help our treatment. That said, from here on out we will refer to anatomic dead space and alveolar dead space in an effort to be more specific with our discussion.



Last type of dead space is what we will call mechanical dead space. Mechanical dead space, which may also be noted as equipment or apparatus dead space, is the dead space that we add on to the system with our devices: vent circuit, end-tidal carbon dioxide ($EtCO_2$) detector, heat & moisture exchanger (HME) or humidifier, etc.[25] To be a bit more specific, it refers to all the things from where anatomic dead space starts (oropharynx/ nasopharynx) to where exhaled air leaves the wye of the vent circuit:



[24] Johnston, 2015 - This article reviews techniques by which we can measure physiologic dead space and then use it to determine a value for anatomic dead space

[25] All of these components get discussed in **Ventilator Anatomy**; heat & moisture exchangers will be discussed further when we get to **Humidifiers**

Mechanical dead space is a problem because it increases the amount of used-up air with which incoming air must be mixed before it gets to the alveoli. In the normal human being, fresh air is pulled into the airways starting right at that imaginary blue line in the above picture; in the ventilated patient, fresh air begins at that wye. We've discussed this effect in the **Appendix**, but suffice it to say that we should try to minimize mechanical dead space when possible (i.e., think about whether or not an in-line suction device or humidifier is needed rather than placing them blindly for all patients) and that the effect is more pronounced with smaller patients and higher respiratory rates (i.e., pediatrics).

One last thing about this is that there is a silver lining to our concept of mechanical dead space. The endotracheal tube actually creates a narrow passageway from the teeth/ lips (where we drew that blue line) down to the trachea, essentially negating the dead space of the naso- and oro-pharynx. So while the net change in overall dead space may be negligible as far as amount added versus amount taken away, we still want to maximize efficiency of ventilation and minimize unnecessary things in our vent circuit when possible.

There is another related concept to consider in this discussion of dead space that doesn't quite fit any of the types mentioned above. We like to think of all of these volumes as fixed quantities of air, but the truth is that the containers that hold this air are flexible or have stretch and therefore we sometimes see differences in expected versus actual values. One example of this is that the amount of air we put into the system doesn't always match up exactly with air out of the system.[26] So where does that air go? Some of it stays in the alveoli (see upcoming discussion on recruitment), some of it leaks around our cuff, some of it is lost to the tissues and airway structures, etc. While this isn't exactly dead space per se, it helps to recognize that it is a thing that can cloud our understanding of air volumes.

Another place where this comes into play is with the vent circuits themselves. These plastic tubes are not rigid and do have a certain amount of stretch to them. If we look at the package of the tubing, there is a value that says how much volume of stretch a given circuit has per unit of pressure. We will revisit this idea in later sections once we discuss a few of the concepts mentioned here, but know that in certain types of ventilation we may inadvertently overestimate the amount of air delivered if we ignore the stretch of the circuit. This is particularly relevant with little patients (i.e., infants), as the impact of this effect (ratio of misestimation to potential outcome) is more pronounced with smaller breaths.[27]

Hypoxic Pulmonary Vasoconstriction[28]

Hypoxia results in vasoconstriction of the pulmonary vascular bed (thus the term hypoxic pulmonary vasoconstriction), which is opposite of what happens in systemic circulation. This mechanism helps the lungs avoid wasting blood supply to part of the lung that isn't getting enough oxygen – it's a mechanism to conserve resources and maximize efficiency in the system. Just as with other vascular beds in the body, the pulmonary capillaries are in a constant state of flux and respond to the needs of the body and the availability of resources (oxygen, in this case, being the driving force) by opening and closing.

Carrying on this conversation with a new term: hypoxic pulmonary vasoconstriction helps to avoid ventilation/ perfusion (V/Q) mismatch, which could look like either of the following:[29]

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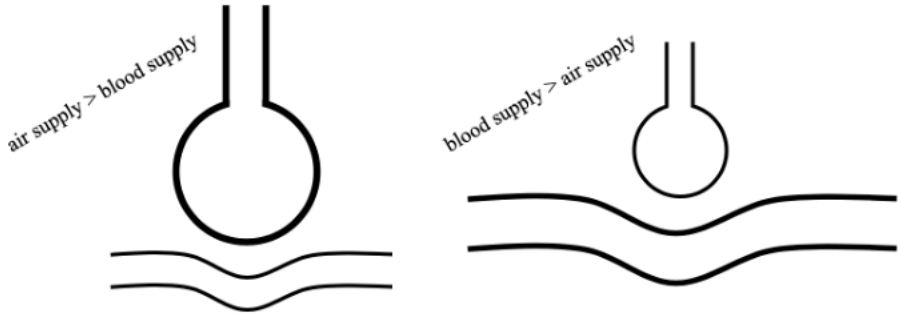
[26] This difference between tidal volume and exhaled tidal volume will be addressed in **Volume Control** and then expanded on in **Tidal Volume**

[27] Bauer, 2018 - He discusses this idea of stretch in the vent circuit in his book on vent management; we also mention it in our discussion of **Volume Control** and then demonstrate this impact in the context of managing a pediatric patient later on in the **Appendix**

[28] For more reading on the subject of hypoxic pulmonary vasoconstriction:
Dunham-Snary & friends, 2017 - They describe how this response can be inhibited by certain interventions and discuss the role of hypoxic pulmonary vasoconstriction in different pathologies
Lumb & Slinger, 2015 - This paper outlines the timelines discussed and also points out a number of relevant pharmacologic agents that contribute to the effect

[29] Mason, 2019 - We just left out the idea of ventilation/ perfusion (V/Q) ratio in this discussion because our focus is on the general idea only, but take a look here for a quick explanation



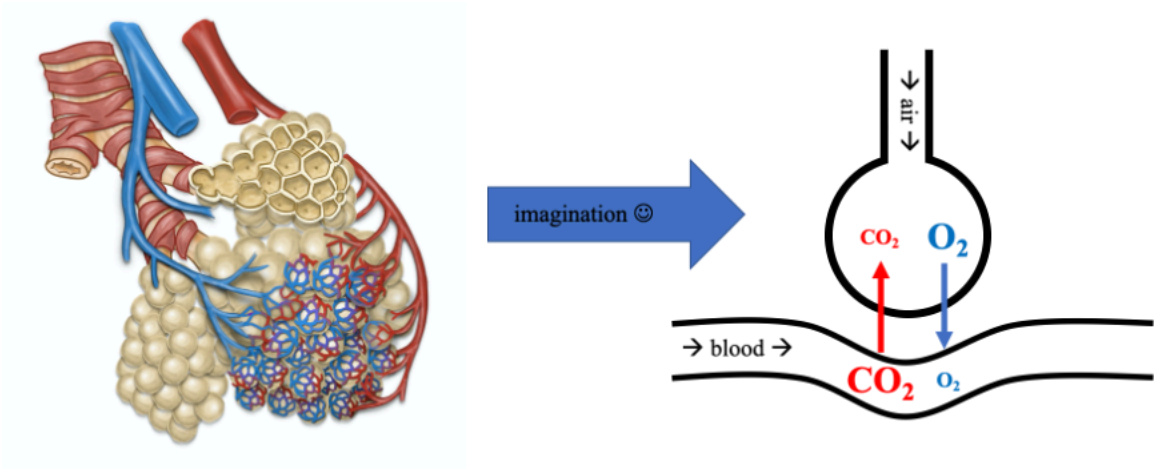
The left side type of ventilation/ perfusion (V/Q) mismatch demonstrates alveolar dead space. It shows that air supply (i.e., oxygen) in the alveolus is in excess of blood flow and therefore some of that oxygen won't get utilized or move into the bloodstream. The right side state is what we call a shunt. In a shunt, blood ends up passing through the pulmonary vascular bed without getting its full complement of oxygen. It isn't always the case that the mismatch is due to volume of air in the alveoli as shown, it can also be related to some kind of impediment that prevents the movement of air into the alveoli. Examples of this would be pulmonary edema, ARDS, and pneumonia. In either of these cases, dead space or shunt, hypoxic pulmonary vasoconstriction is one of the body's mechanisms for either reversing or avoiding these types of mismatches.

Now one thing to know about this whole process is that it goes both ways: vasoconstriction is the response to hypoxia and vasodilation occurs when oxygenation is adequate or that hypoxic state is resolved. We might consider these to be similar processes, just in opposite directions. There is a distinction, however, in the rate at which either change happens. The initial hypoxic vasoconstriction side of things happens on the order of seconds to minutes; the reverse process (vasodilation) typically also occurs quickly, but can happen much more slowly (up to hours) or incompletely (without complete reversal of the vasoconstriction) when the hypoxic pulmonary vasoconstriction response has been sustained for a while.

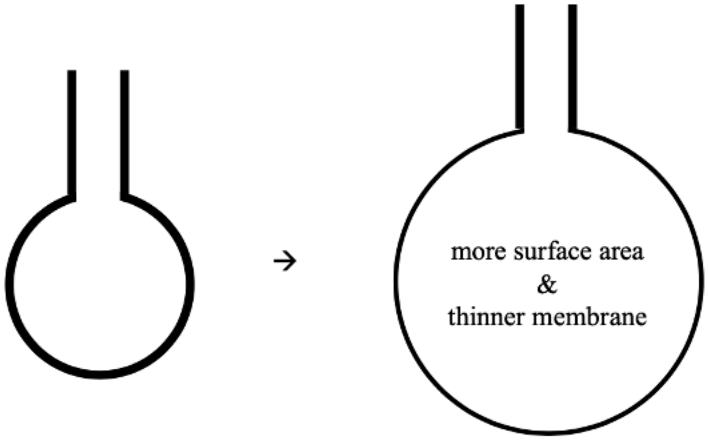
The fact that it may take quite some time to reverse this process helps to explain, in part, why we aren't always able to fix our ventilated patients as well as we want to in the short span we get to hang with them in transport. It also helps bring out the idea that just because a patient doesn't look awesome when we get there doesn't mean that the sending facility or crew has been doing things wrong – they may be taking the right steps and called us before enough time has passed for the fix to manifest. There are many more intricacies and effects of hypoxic pulmonary vasoconstriction on the body (see all those references on the previous page), but the main point is that we may not be able to fix a super sick patient quickly. And that's just fine. We do what we can (as we will outline soon) and recognize that there are limits to the results we can expect.

Alveolar Surface Area

Even though we have been demonstrating the alveoli-capillary interface as a single blood vessel running past an air sac, it is important to recognize, again, that this is a simplification of how things are and that the surface of the alveoli is covered by a network of vessels:[30]

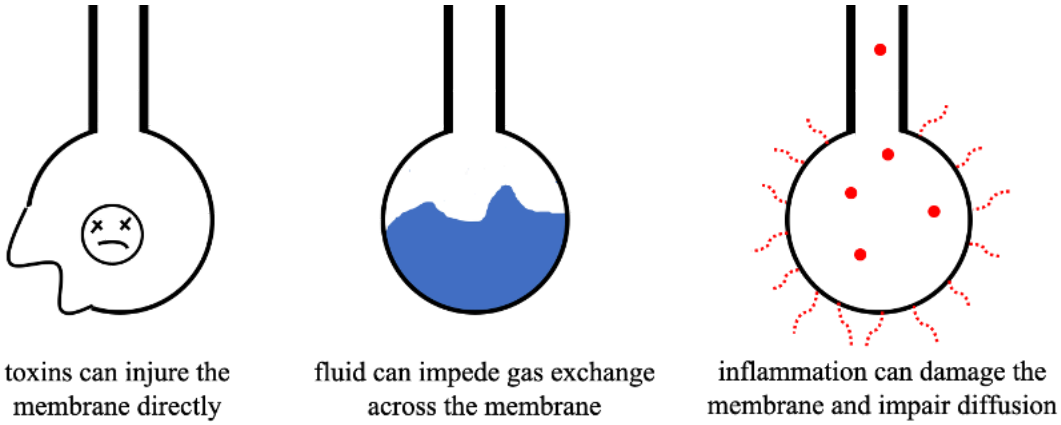


When we inflate the alveoli we get more surface area and that means more interface between air and blood. In addition, inflation of the alveoli causes the alveolar membrane to stretch and become thinner, allowing for easier diffusion of gases:[31]



[30] Left bit of this graphic built by Dr. Sameer of *Art by Dr. Sam* on Fiverr and *The Young Orthopod* on YouTube
[31] Desai, 2012 - We spell out this diffusion process in much more detail in the section on **Oxygenation**, but here's a video to explain how these factors are related to gas exchange across the alveolar-capillary membrane

More surface area and a thinner membrane make it easier to move air from inside of the alveoli to the circulatory system, so lots of our interventions with the vent are focused on this idea. That said, certain things can get in the way of this gas exchange even if we do get the surface area up and the membrane thinned out. Think of these as things that impact access to usable alveolar surface area:[32]



Now there are ways to work through these impediments and we'll talk about strategies to make that happen.[33] Also know that while more alveolar surface is important, if taken too far it can cause overinflation injury (barotrauma or volutrauma) which results in damage to this membrane.[34] How we maintain safety of the alveoli is something we'll get to later on.[35] For now we'll summarize it like this: management of the alveolar surface is important for gas exchange and there are both interventions we can take to improve the efficiency of gas diffusion across that membrane and steps to take to maintain safety and avoid causing further damage.

Ideal Body Weight

Second to last thing before we move on to talking about the machine: lung size is most strongly correlated with patient height. Because of this, we use a patient's height to calculate an ideal body weight (IBW) when doing vent things.[36] The idea is that a six-foot human could weigh either 120lbs or 300lbs and the size of his or her lungs wouldn't change. This is critical in both directions: we don't want to cause injury by pushing too much air into smaller lungs, we also don't want to underventilate someone who is simply underweight.

There is a formula to calculate IBW for both males and females, often presented as a hybrid of metric and standard units:

$$IBW_{male}\ (kg) = 2.3(\text{height in inches} - 60) + 50$$
$$IBW_{female}\ (kg) = 2.3(\text{height in inches} - 60) + 45.5$$

[32] George, 2015 - Check this out for a bit of extra detail on the difference between pneumonia and pneumonitis, both of which would be included in this working list of things that can inhibit effective gas exchange
[33] Strategies to facilitate gas exchange are discussed in the section on **Oxygenation**
[34] Yartsev, 2025d - Further reading on barotrauma, volutrauma and other types of injury related to mechanical ventilation; while the terms are often used interchangeably, they are different concepts
[35] We talk about ensuring alveolar safety in many places in this manual, but mostly in the sections on **Control of Ventilation** and **Plateau Pressure**
[36] IBW may also be referred to as predicted body weight (PBW)

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For the metric enthusiasts, we also have it as so:

$$IBW_{male}\ (kg) = 0.91(\text{height in cm} - 152.4) + 50$$
$$IBW_{female}\ (kg) = 0.91(\text{height in cm} - 152.4) + 45.5$$

Or we can use charts like this:[37]

HEIGHT	PBW	4 ml	5 ml	6 ml	7 ml	8 ml
4' 0" (48)	17.9	72	90	107	125	143
4' 1" (49)	20.2	81	101	121	141	162
4' 2" (50)	22.5	90	113	135	158	180
4' 3" (51)	24.8	99	124	149	174	198
4' 4" (52)	27.1	108	136	163	190	217
4' 5" (53)	29.4	118	147	176	206	235
4' 6" (54)	31.7	127	159	190	222	254
4' 7" (55)	34	136	170	204	238	272
4' 8" (56)	36.3	145	182	218	254	290
4' 9" (57)	38.6	154	193	232	270	309
4' 10" (58)	40.9	164	205	245	286	327
4' 11" (59)	43.2	173	216	259	302	346
5' 0" (60)	45.5	182	228	273	319	364
5' 1" (61)	47.8	191	239	287	335	382
5' 2" (62)	50.1	200	251	301	351	401
5' 3" (63)	52.4	210	262	314	367	419
5' 4" (64)	54.7	219	274	328	383	438
5' 5" (65)	57	228	285	342	399	456
5' 6" (66)	59.3	237	297	356	415	474
5' 7" (67)	61.6	246	308	370	431	493
5' 8" (68)	63.9	256	320	383	447	511
5' 9" (69)	66.2	265	331	397	463	530
5' 10" (70)	68.5	274	343	411	480	548
5' 11" (71)	70.8	283	354	425	496	566
6' 0" (72)	73.1	292	366	439	512	585
6' 1" (73)	75.4	302	377	452	528	603
6' 2" (74)	77.7	311	389	466	544	622
6' 3" (75)	80	320	400	480	560	640
6' 4" (76)	82.3	329	412	494	576	658
6' 5" (77)	84.6	338	423	508	592	677
6' 6" (78)	86.9	348	435	521	608	695
6' 7" (79)	89.2	357	446	535	624	714
6' 8" (80)	91.5	366	458	549	641	732
6' 9" (81)	93.8	375	469	563	657	750
6' 10" (82)	96.1	384	481	577	673	769
6' 11" (83)	98.4	394	492	590	689	787
7' 0" (84)	100.7	403	504	604	705	806

PBW and Tidal Volume for Females

ARDSNet Studies

HEIGHT	PBW	4 ml	5 ml	6 ml	7 ml	8 ml
4' 0" (48)	22.4	90	112	134	157	179
4' 1" (49)	24.7	99	124	148	173	198
4' 2" (50)	27	108	135	162	189	216
4' 3" (51)	29.3	117	147	176	205	234
4' 4" (52)	31.6	126	158	190	221	253
4' 5" (53)	33.9	136	170	203	237	271
4' 6" (54)	36.2	145	181	217	253	290
4' 7" (55)	38.5	154	193	231	270	308
4' 8" (56)	40.8	163	204	245	286	326
4' 9" (57)	43.1	172	216	259	302	345
4' 10" (58)	45.4	182	227	272	318	363
4' 11" (59)	47.7	191	239	286	334	382
5' 0" (60)	50	200	250	300	350	400
5' 1" (61)	52.3	209	262	314	366	418
5' 2" (62)	54.6	218	273	328	382	437
5' 3" (63)	56.9	228	285	341	398	455
5' 4" (64)	59.2	237	296	355	414	474
5' 5" (65)	61.5	246	308	369	431	492
5' 6" (66)	63.8	255	319	383	447	510
5' 7" (67)	66.1	264	331	397	463	529
5' 8" (68)	68.4	274	342	410	479	547
5' 9" (69)	70.7	283	354	424	495	566
5' 10" (70)	73	292	365	438	511	584
5' 11" (71)	75.3	301	377	452	527	602
6' 0" (72)	77.6	310	388	466	543	621
6' 1" (73)	79.9	320	400	479	559	639
6' 2" (74)	82.2	329	411	493	575	658
6' 3" (75)	84.5	338	423	507	592	676
6' 4" (76)	86.8	347	434	521	608	694
6' 5" (77)	89.1	356	446	535	624	713
6' 6" (78)	91.4	366	457	548	640	731
6' 7" (79)	93.7	375	469	562	656	750
6' 8" (80)	96	384	480	576	672	768
6' 9" (81)	98.3	393	492	590	688	786
6' 10" (82)	100.6	402	503	604	704	805
6' 11" (83)	102.9	412	515	617	720	823
7' 0" (84)	105.2	421	526	631	736	842

PBW and Tidal Volume for Males

ARDSNet Studies

Some people remember this formula for ideal body weight as "inches over five feet" as shown below. The only problem with this is that it gets tricky if we have someone under five feet, but either way works:

$$IBW_{male}\ (kg) = 2.3(\text{every inch over 5'}) + 50$$
$$IBW_{female}\ (kg) = 2.3(\text{every inch over 5'}) + 45.5$$

When dealing with pediatric patients, our go-to reference is often the Broselow Tape, but we do have some formulas we can refer to if that isn't available:[38]

Infant Weight (kg) = 0.5(age in months) + 4
Little Kid (1 – 4 years) Weight (kg) = 2(age in years + 5)
Big Kid (5 – 14 years) Weight (kg) = 4(age in years)

And note that the Broselow overlaps with the equations and chart above, so if we have a small grownup or a big kid we should still be able to get an ideal body weight just fine.

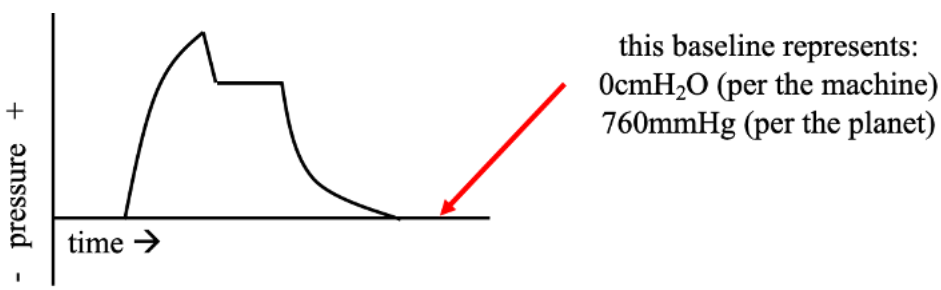
[37] NHLBI ARDS Network, 2005 (image)
[38] Graves & friends, 2014 - There are lots of formulas out there to estimate weights for pediatrics, but we went with recommendations from this paper comparing different methods

Measuring Pressures

During mechanical ventilation pressures are measured in centimeters of water (cmH_2O). We may occasionally hear this pronounced as "sonnimeters" of water and know that a sonnimeter and a centimeter, in this context, are the same thing. So we have cmH_2O with mechanical ventilation, but we generally talk about ambient air pressures in other terms, such as mmHg, kPa, PSI, etc. We skimmed right on past this concept in a previous section when we said that 1mmHg is about 1.4cmH_2O,[39] but let's now put it all down in a chart just to make things clear:[40]

	ATM	PSI	kPa	mmHg	cmH_2O
ATM	1	14.7	101.3	760	1033
PSI	0.068	1	6.89	51.7	70.3
kPa	0.0099	0.145	1	7.5	10.2
mmHg	0.0013	0.019	0.133	1	1.36
cmH_2O	0.00097	0.014	0.098	0.736	1

Also, note that we assume ambient pressure as it relates to airway and vent stuff is zero; so while true atmospheric pressure at sea level is 760mmHg (1 ATM), we call it 0cmH_2O to make things easier.[41] And then we have a way to represent breaths we give as waveforms showing pressure as a function of time with this new zero point (representing atmospheric pressure) as the baseline. For now we are going to ignore **Positive End-Expiratory Pressure** (since we haven't discussed it yet); we also don't have to worry about the specific components of the waveform – all those things will be discussed later on:[42]



[39] This was in **How is Positive-Pressure Ventilation Different?** when we were talking about the fact that a negative-pressure, spontaneous breath only takes -1mmHg of pull while a typical positive-pressure breath via the machine takes 15-25cmH_2O to move an equivalent amount of air

[40] We built this chart by Googling conversions for these values…

[41] Yartsev, 2021b - Scroll down to the section called *Airway Pressure* for some interesting background on why we measure and label pressures the way we do

[42] Know that these waveforms are a bit exaggerated for demonstration purposes, we'll discuss actual waveforms in **Vent Graphics**

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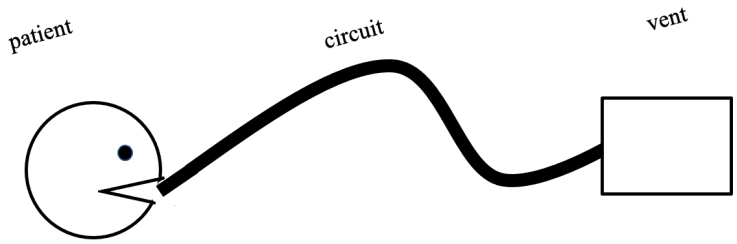
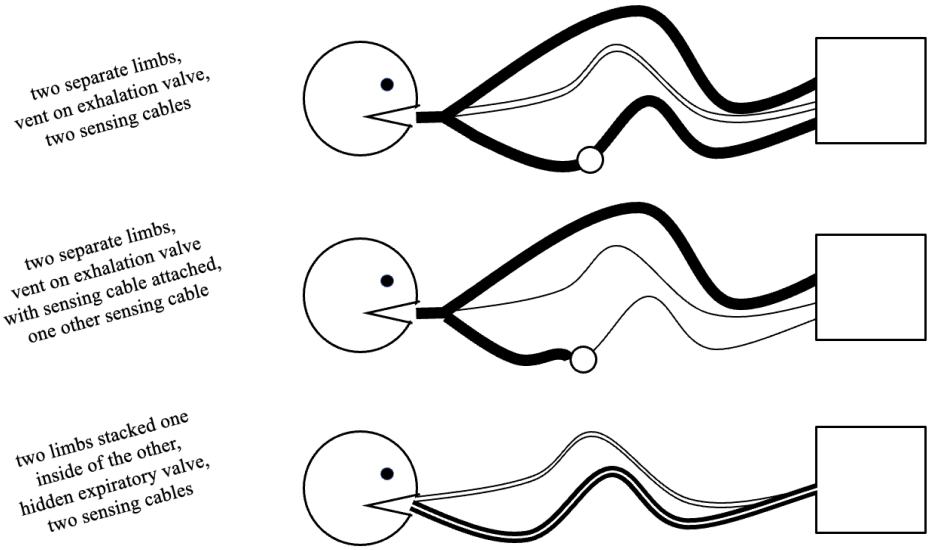
Ventilator Anatomy

Before we dive into the specifics about how the vent works and what settings we might choose in different situations, let's outline how the machine itself is set up and what each of the components are. At its most basic, the vent is attached to the patient via a circuit:

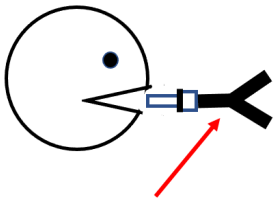


The circuit itself is generally composed of two distinct tracks or limbs, one for inhalation and one for exhalation. On the exhalation side, there may be an obvious opening through which exhaled air passes. There are also a few hoses that run alongside the circuit, these are used to sample the air involved - most commonly movement (flow) or pressure of specific gases. Here are a few examples of what a circuit might look like:



There is a bit of variation between models, but they all contain more or less the same components.

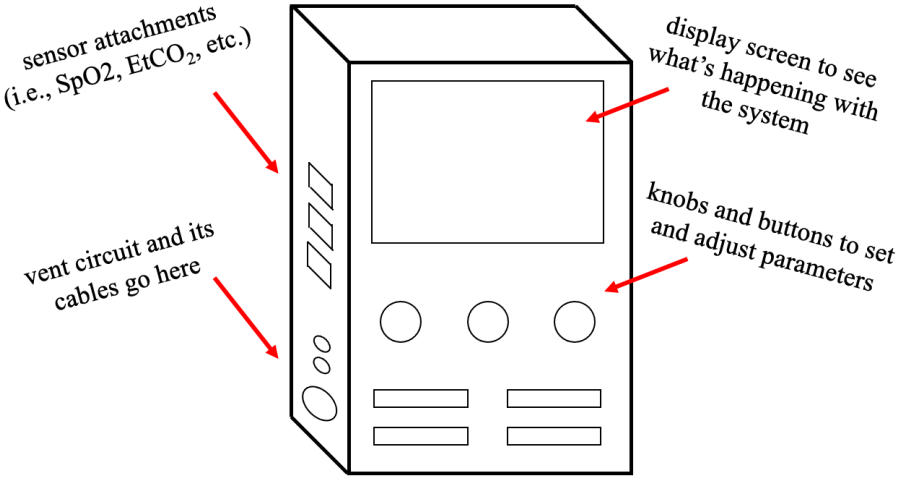
One landmark to point out is what is often referred to as the wye (or simply the "y"). This is where the inspiratory and expiratory limbs split close to where the circuit attaches to the endotracheal tube:



Some vent circuits are designed so that the two limbs of the circuit are stacked on within the other so that it looks like a single tube. Think of this "coaxial" setup as two pipes, one running inside the other. The wye in this type of circuit is still at the same point - just distal to the endotracheal tube - we just can't see it as clearly (i.e., it doesn't look like a "y").

One last thing to mention about circuits before we get back to the machine itself: it is quite common that things are inserted into the circuit during clinical care. Examples would be **Filters**, **Humidifiers** devices to deliver **In-Line Nebulization** or suction devices (i.e., a ballard).

And then on the machine itself there are a few sections: one where we attach all the things, another where we press buttons to input and change settings, and then likely one more where we can see or visualize what feedback the machine is getting from its sensors. This is just one hypothetical example:



On the screen itself, quantitative values are often grouped in a way that makes it easy for the clinician to identify information. It is quite common that measured parameters are grouped independently or separate from settings which are chosen by the operator. And then less-clinically-relevant settings may not live on the home screen at all, rather they are likely buried in menus as they don't always need to be referenced quickly or at a glance. And then some machines allow the user to choose which things get displayed on the home screen.

One thing to mention here: not all vents have all of the features and even if a particular machine has a feature, that does not necessarily mean it has to be utilized. One example of this is pulse oximetry and capnography - while some transport ventilators are able to monitor and display this information, it may (and often does) make more sense to monitor these things through another device, such as a cardiac monitor, and forgo using that feature on the vent.

With that quick overview of components, let's move on to our discussion of ventilator management.

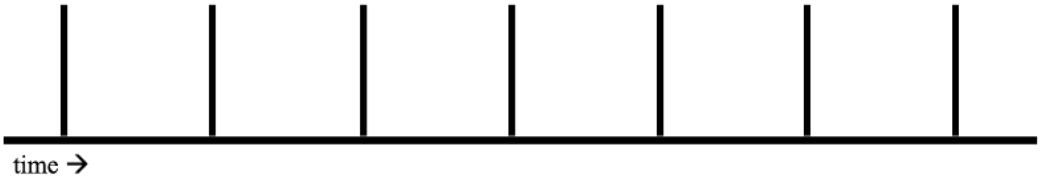
Modes of Ventilation

This next section discusses how we organize the delivery of breaths to a patient. We've distinguished this concept of mode with that of control (see next section) in order to make things easier to conceptualize, but the terms sometimes get used with a bit of overlap. In reality, both the way we control breaths and how we organize those breaths are components of how we describe what mode we are in. While we could alternatively label this idea (what we refer to here as modes of ventilation) as breath sequence, the convention in transport is to identify this idea using the term mode, so we'll stick with that. Moving forward, we will refer to mode as the overall pattern or organization of breaths and control as the specific way we choose to deliver them.[43]

Before we get there, however, there is one concept that will be needed in order to understand things in the next few sections. **Triggers** are the thresholds by which the machine knows when to give a breath. We will talk about these in detail later, but it is tough to explain the following ideas without a basic comprehension. In its simplest form, a trigger could simply be a quantity of time. An example of this would be one breath given every four seconds; we could then describe those instances as time-triggered breaths. There are also ways the vent can infer inspiratory effort made by the patient and give breaths based on those cues. We'll discuss how that happens later, but the general term for this type of thing is a patient trigger (i.e., a patient-triggered breath) and we will use that idea shortly.

Basic Modes of Ventilation

The simplest way to ventilate a patient with a machine is to give breaths on a schedule and to ignore what the patient does on his or her own. Let's assume a hypothetical timeline running left to right over an arbitrary amount of time with black hashes to represent time-triggered breaths:



Now if the patient tries to take breaths overtop of this timeline, that effort gets ignored. We'll show it like this:

In terms of triggers, we could say that ventilation via this mechanism utilizes time triggers only and does not have a mechanism for patient triggers. Now there are two versions of this type of ventilation, controlled mechanical ventilation and intermittent mandatory ventilation (IMV).[44] The difference here is subtle: in controlled mechanical ventilation (which historically precedes all the other modes), the patient is physically unable to draw a breath on his or her own with effort made; in intermittent mandatory ventilation (a subsequent

[43] Chatburn & friends, 2014 - For specifics on how all of these modes and concepts of ventilation ought to be labeled and described, this article outlines a taxonomy for vent things

[44] Frakes, 2007; Kacmarek & Branson, 2016 - We won't refer to controlled mechanical ventilation as CMV (even though we sometimes see it that way), because CMV more often refers to continuous mandatory ventilation which we describe in just a moment; for more on this refer to both articles referenced

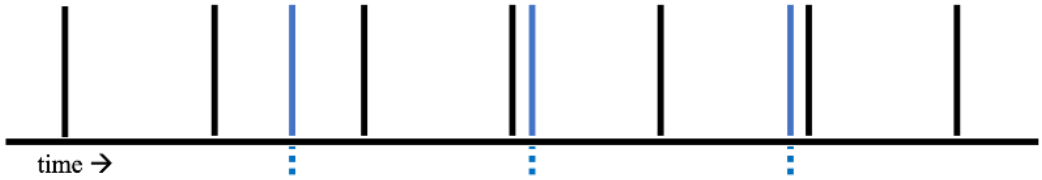
technological improvement) the patient is, in fact, able to draw a breath, it's just that the machine doesn't offer support and instead allows the patient to draw in air from the vent circuit independently and without help.

Controlled mechanical ventilation isn't routinely used, but it does get utilized in surgical settings when patients are paralyzed and for sure not making an effort to breathe spontaneously. Intermittent mandatory ventilation, on the other hand, is normally not available on its own – it typically comes with additional features which we will line out in just a bit.[45] Now we may be able to manipulate our vent to approximate either of these modes in the event that we want to ignore patient triggers altogether, but those would be specific cases and we'll get to them later on.[46]

Moving on, subsequent modes build upon this framework by introducing mechanisms to support a patient's effort to breathe. When we introduce patient triggers, however, there is the potential that harm can result and we will talk about how different modes work to mitigate that risk. That said, utilizing the machine to augment patient effort improves comfort, facilitates recovery, reduces the negative effects of positive-pressure ventilation, and gives us more control over the management of the patient.[47] So now let's get on to the two modes commonly used in transport that take us beyond the basics and allow us to use patient triggers.

Assist Control

Assist control (AC), also known as continuous mandatory ventilation (CMV), is a mode that augments a patient's spontaneous respiratory effort by delivering a preset amount of air when an inspiratory effort is detected. To describe it in terms of triggers: time-triggered breaths are given on a schedule, but patient-triggered breaths can also occur in addition to those. Here's how it would look mapped out graphically:

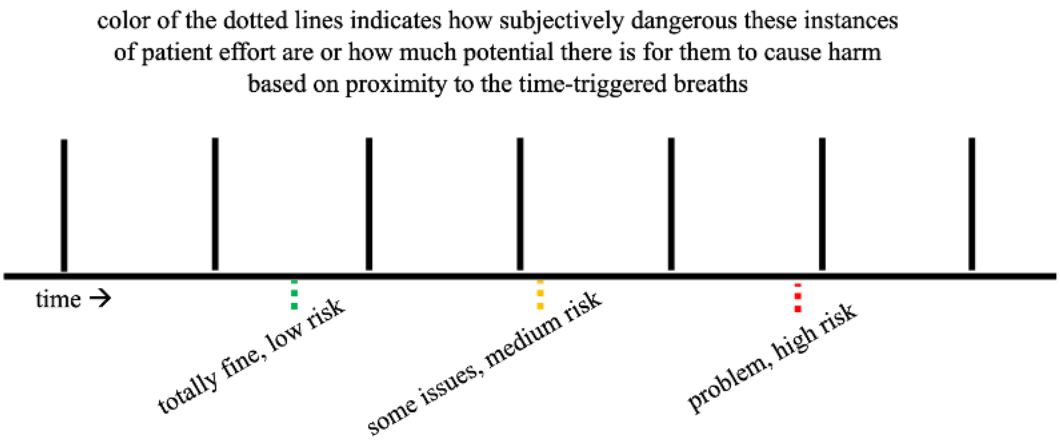


[45] Discussed in **Synchronized Intermittent Mandatory Ventilation**

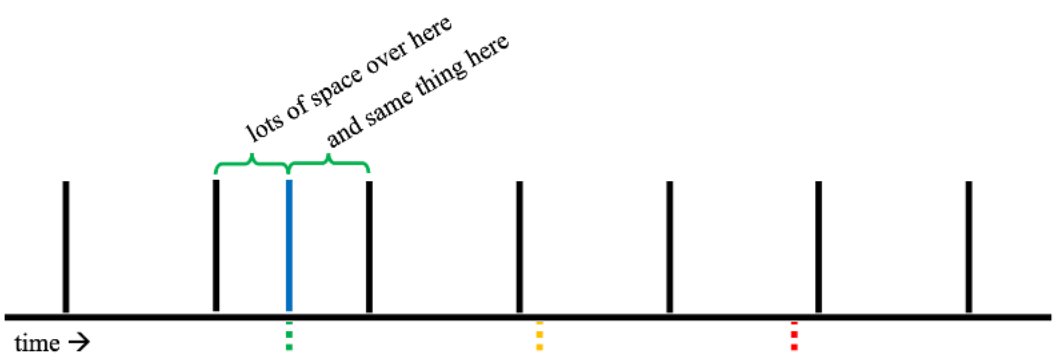
[46] These cases in which we might want to ignore patient-triggers are discussed in both **Overbreathing** and **Acute Deterioration**

[47] Mauri & friends, 2017 - We will talk about the specific ways in which patient-triggered breaths are of benefit later on (in **Comfort**), but the article provides a bit of context for these claims

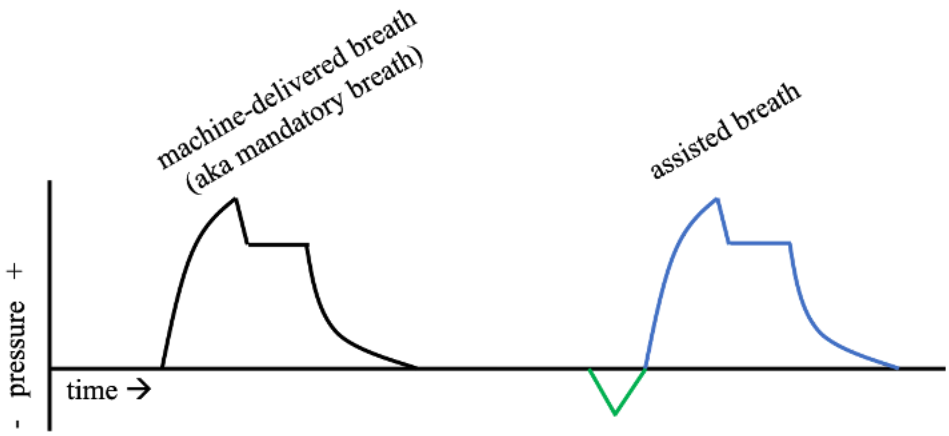
In assist control, all breaths are the same size. This means that patient-triggered breaths are the same (in terms of volume or pressure, see upcoming sections) as the machine-delivered, time-triggered ones. The obvious advantage here is that the patient's expressed need for more air per unit time is readily met. On the other hand, this method of giving full breaths whenever a trigger is sensed has the potential to cause some harm. To start to make sense of this, let's return back to a series of time-triggered breaths with effort notated by dotted lines below the timeline. In assist control, each instance of patient effort (whether true patient effort or simply perceived patient effort due to some other factor) has a varying potential for harm based on where it lines up in relation to other breaths. We'll draw it out first and then explain in detail:



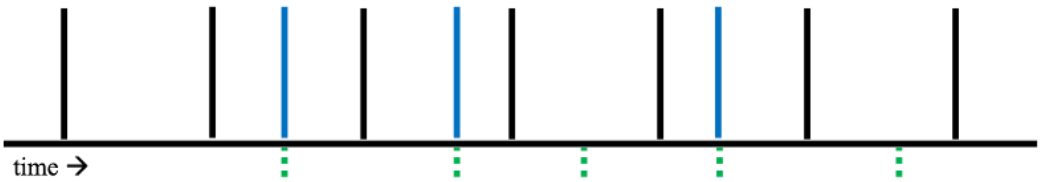
To expand on this: with the green effort, there is space (in time) on either side of the breath, so the machine can actualize that breath without affecting other breaths in proximity:



The difference between that green, patient-triggered breath and the baseline, time-triggered ones can be represented via one of those pressure-over-time waveforms that we mentioned before. Note the dip at the start of the second waveform as the patient breathes in – this is the effort that gets sensed by the machine right before a full positive-pressure breath is then given:[48]



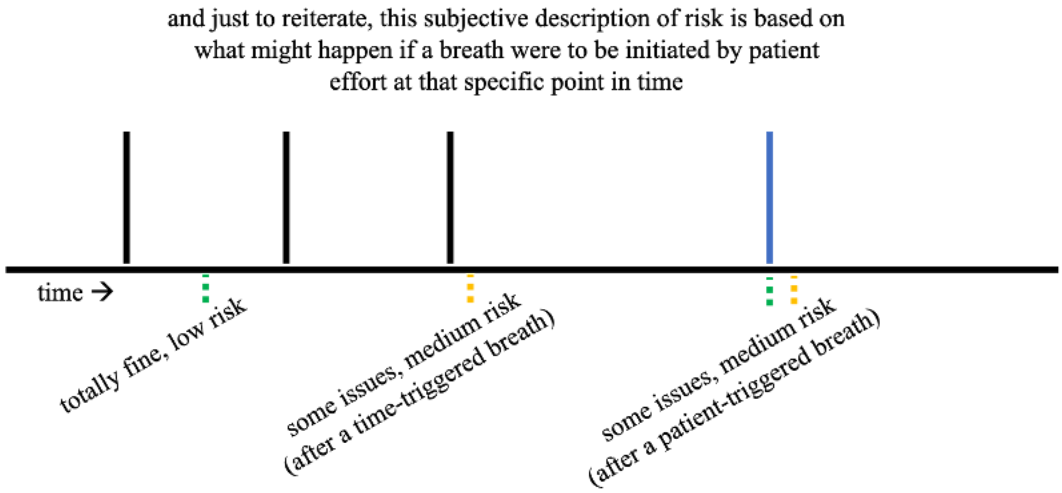
A pretty good assist control situation might look something like this where the patient's need for more breaths is generally met and that need, in the form of inspiratory effort, doesn't interfere or overlap with the scheduled breaths:



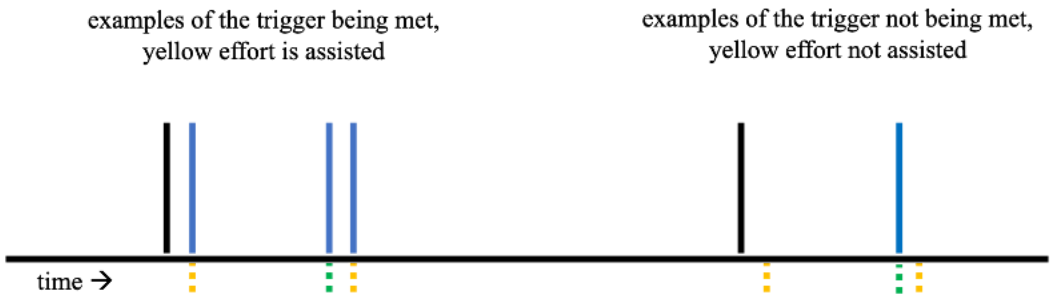
Do note that not all patient effort will result in delivered breaths. That's where triggers come in and we can adjust that parameter to make it more or less likely that a breath will occur. Again, more on this later, just something to note for now. And the ideal assist control situation (in contrast to the pretty good one shown above) would be when all patient effort results in a delivered breath, none of those patient-triggered breaths interfere with the time-triggered ones, and when no miscellaneous factors cause accidental triggers. No need to draw that one out, as it will be much clearer later on. For now we want to stay on track with describing assist control and how we can potentially cause harm in this mode.

[48] Now this graphic makes it seem as if a pressure change detected by the machine leads to an assisted breath; while that could potentially be the case, the more common situation is a flow trigger (see **Triggers** for more)

In the case of the yellow effort (which we labeled as medium risk) there is a potential trigger that immediately follows another breath. It could be that the other breath in question is a machine-delivered one (as we had it in the first graphic) or it could be another patient-triggered one:

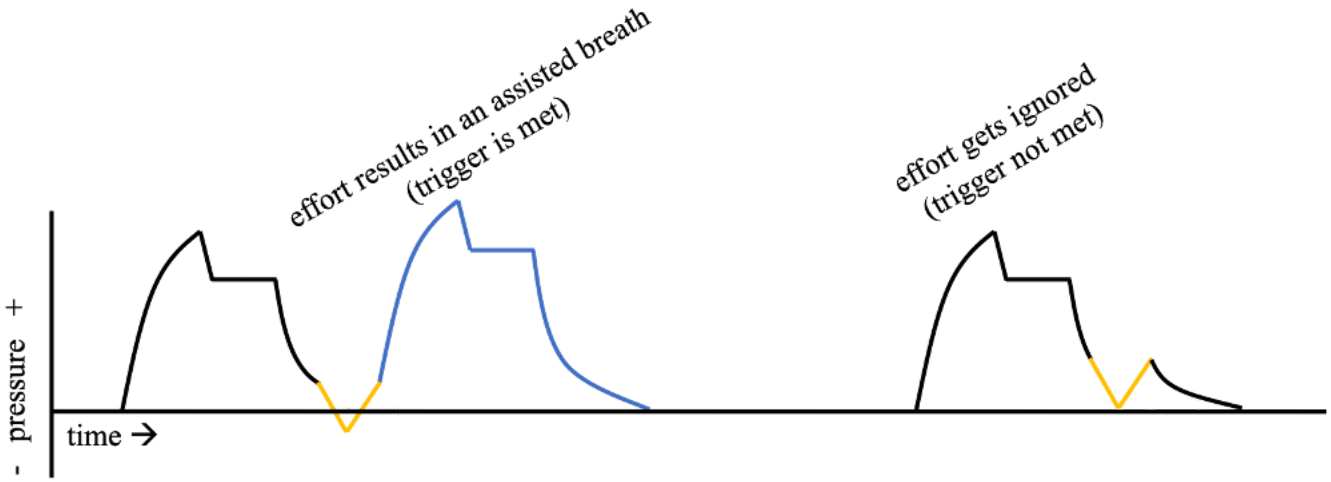


Now what happens next depends largely on how the trigger is set up, but we can generalize it by saying that the further along the first breath is or the closer the breath is to an end-exhalation baseline, the more likely that the effort will catch and result in a full breath. There are two possible outcomes: one in which the trigger results in an assisted breath and one in which the trigger does not result in a breath:[49]

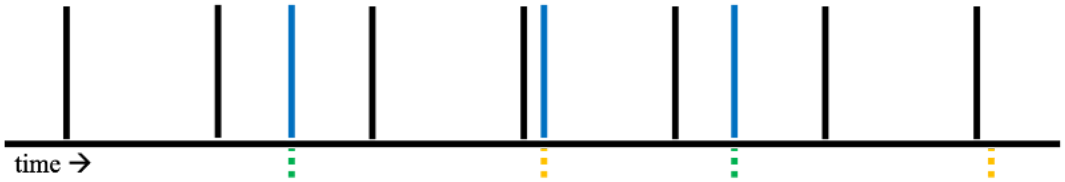


[49] And as already mentioned, we will discuss this idea of **Triggers** in much more detail later; also framing this situation as a binary thing with only two outcomes is an oversimplification, the nuance of what happens when patient effort occurs at various points along a delivered breath is discussed and illustration in **Vent Graphics**

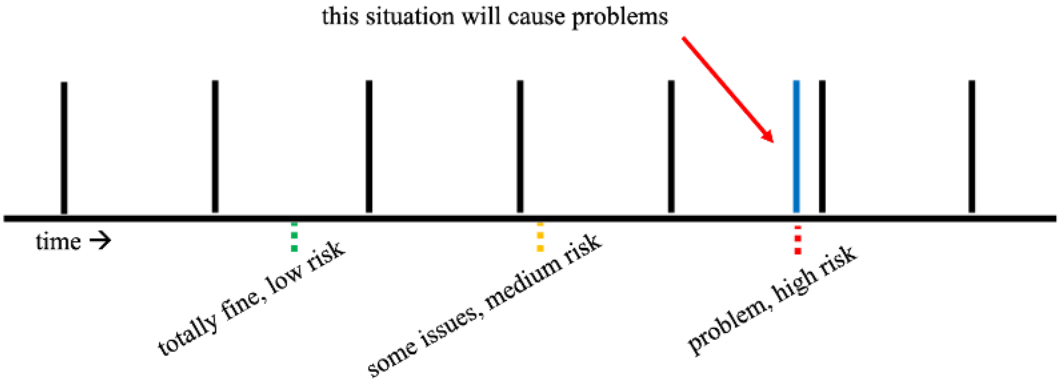
And then we can carry on with the idea to show either of those outcomes as a waveform of pressure over time:



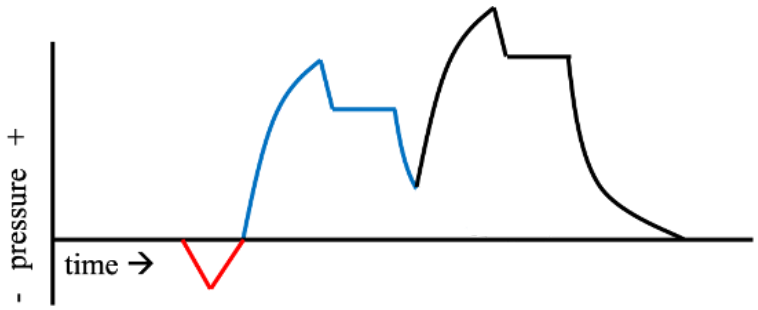
These yellow-effort situations do have the potential to cause harm, but they are less likely to result in breaths being delivered because of the ongoing breath that precedes them. That harm comes from the increased pressure as a full breath is given before the prior one was completely done (left side, note the drift of maximum height on waveform). If the yellow effort doesn't result in a breath being delivered, this may cause some discomfort (right side, due to an expressed need that goes unaddressed), but that's probably OK. That said, a combination of green and yellow effort is just fine for our patients in assist control:



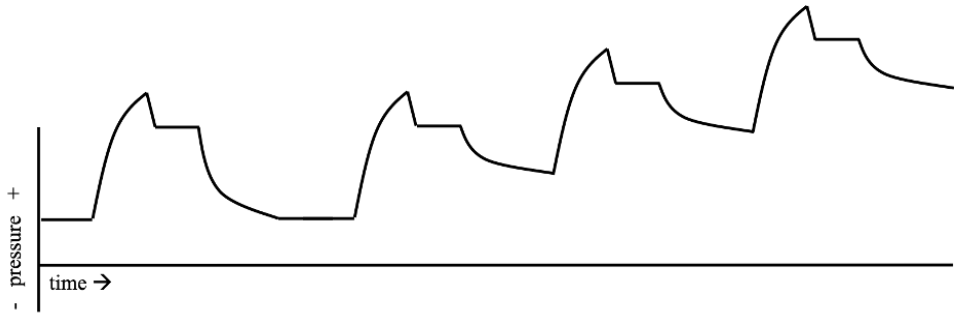
The issues with assist control begin when we get those red-effort situations in which a patient-triggered breath immediately precedes a time-triggered one:



When this happens we get that same increase in pressure we just described (the drift of maximum height on the pressure waveform), but consistently and to a greater effect. As a waveform it would look like this:



There is a complication known as **AutoPEEP**, or intrinsic positive end-expiratory pressure, in which this happens with some regularity resulting in sustained high pressures.[50] AutoPEEP can also occur in assist control if we trigger a number of breaths in sequence. We'll revisit this idea again later, but here's how that might look (and we've left out colors and triggers for clarity):[51]



It is worth noting that there are some ventilators designed to avoid this sort of high-risk situation. Many hospital ventilators currently in use and some transport ventilators have a mechanism by which the machine will sense a patient-triggered breath that precedes a machine-delivered breath and, in response, will withhold that second breath in order to avoid potential problems. Think of it as a refractory period in which another breath cannot be delivered.[52]

So we've established that AutoPEEP can occur in assist control, but recognize that it doesn't always happen and that we can take proactive steps to make the mode safe for our patients. The primary benefit of assist control mode is that we can readily meet the patient's need for more volume using patient-triggered breaths. Another benefit is that it is predictable: patient effort that meets the trigger will get assisted to whatever parameter we have set into the machine. To say it another way, time-triggered and patient-triggered breaths will be the same. And then subjectively, assist control is easy to use. While this may not be the best reason to advocate its use in the field, it is simple to set up, easy to conceptualize, and when the primary complication of the mode does arise (AutoPEEP) there are specific actions we can take to fix it.[53]

To summarize, assist control delivers time-triggered breaths at a set rate and will supplement that with full breaths whenever a patient effort meets the trigger threshold. Upside to is that the increased needs of the patient are readily met, downsides are the risk for increased pressures and a move away from baseline (AutoPEEP, which we will get to later). As a general rule: anytime we have someone in assist control we need to be vigilant and monitor both airway pressures and AutoPEEP.

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[50] PEEP or **Positive End-Expiratory Pressure** is discussed later in its own section

[51] In reality these stacked breaths will likely get cut short due to pressure limits we have set on the machine, but we'll explain that fully when we get to the section on **AutoPEEP**

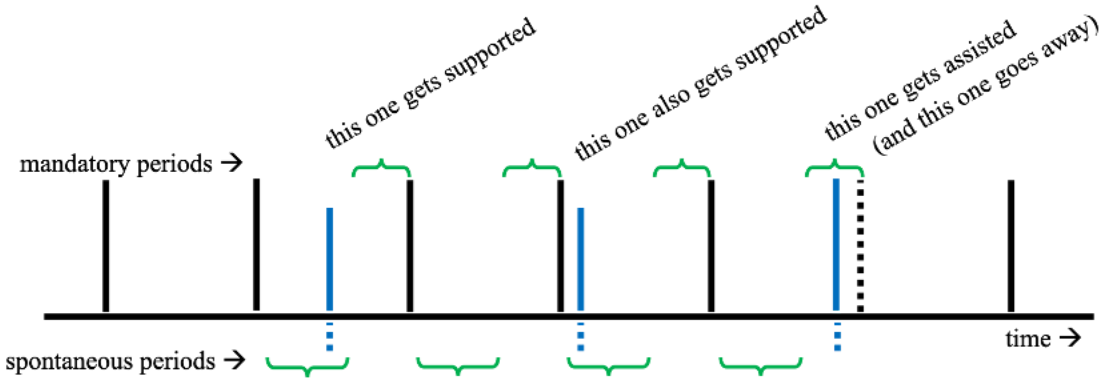
[52] Chatburn & friends, 2014 - This concept is mentioned in this paper on labeling different **Modes of Ventilation**

[53] And the specifics of how we both avoid and fix breath stacking will be discussed later, both in **AutoPEEP** and **Triggers**

Synchronized Intermittent Mandatory Ventilation

Synchronized intermittent mandatory ventilation (SIMV) is an alternative mode of ventilation that seeks to mitigate the shortcomings of the more basic modes by using patient triggers, but has a mechanism built in that helps avoid the **AutoPEEP** complication of **Assist Control**. SIMV starts with the idea of mandatory breaths or a guaranteed number of time-triggered breaths to be given per minute. It then will support breaths taken in between these mandatory breaths. Furthermore, SIMV recognizes when patient effort is made in close proximity to a time-triggered, mandatory breath and assists that effort in a way similar to how breaths were assisted in assist control (i.e., a full breath is given). There are more differences between these various **Types of Breaths** and we'll get back to that eventually, but let's focus on the timing aspect of SIMV first. Going back to our original idea:

SIMV's method for determining how to handle the instances of patient effort is to break the timeline into two alternating categories: mandatory and spontaneous periods. If a patient trigger happens within a spontaneous period, it gets supported and that effort is facilitated by the machine in a manner that we will discuss soon;[54] if an effort occurs within a mandatory period it gets assisted, a full breath is delivered, and the breath that had been planned for that mandatory period gets skipped:[55]



To delve deeper on this graphic, we'll point out a few things. We are assuming that each instance of patient effort meets the trigger threshold to result in a breath. The difference in height of the blue lines is to distinguish supported breaths (smaller, left two) from assisted breaths (taller, right one). And that time-triggered breath at the far right gets omitted to avoid the AutoPEEP complication we saw in assist control.

As for the difference between supported breaths and assisted breaths: supported breaths only get a little bit of help from the machine and the assisted breaths are fully facilitated by the machine to a target amount of air, just as in assist control. Supported breaths are always supported via pressure, which helps the patient draw a breath a little bit easier; assisted breaths are carried out to meet specific goals by the machine based on settings we input and can be either volume-targeted or pressure-targeted (which we will expand on in the next

[54] Ollie, 2015 - This video explains the timing aspect of SIMV and how it was an improvement to intermittent mandatory ventilation (which we mentioned already in **Basic Modes of Ventilation** and will revisit again in just a moment)

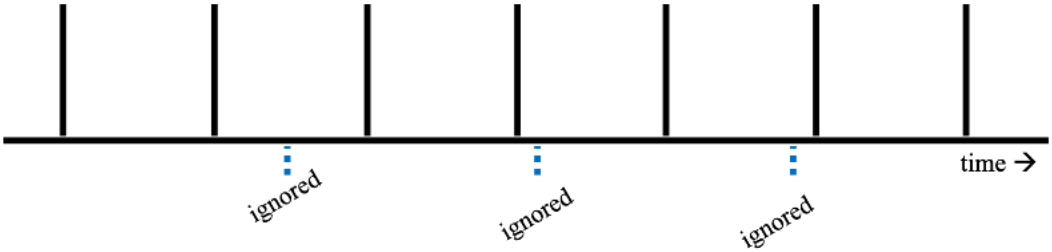
[55] Wheeler & friends, 2008; Kumar, 2015 - The first explains this process of supporting a breath within the mandatory period as we've labeled it, the other is a brief overview that explains it using a different labeling system

section).[56] The practical difference is that pressure support breaths will give us a variable result that depends largely on the patient's contribution to that specific breath, while assisted breaths are more predictable.[57]

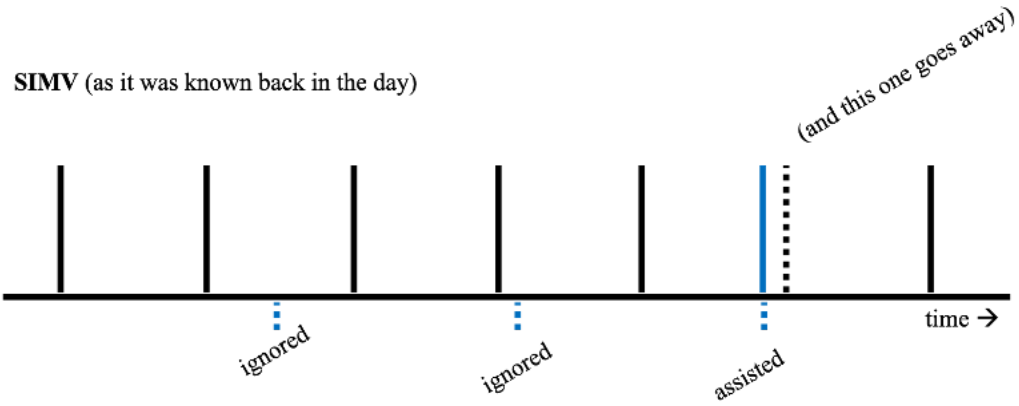
At the risk of getting ahead of ourselves, pressure support breaths are often expected to be less than or smaller than mandatory and assisted breaths (in terms of volume of air). While it may make sense to titrate pressure support up so that supported breaths match the other ones in this regard, it isn't quite as simple as increasing the pressure support value on the machine. That said, there is no reason that the volume of air in a pressure support breath should be less than the other ones, it's more an issue that it often just happens to turn out that way because of the details as to how these different **Types of Breaths** are brought into existence by the machine.[58]

And a few more things about SIMV mode: It originally came onto the scene as intermittent mandatory ventilation, which we already discussed. The "S" for synchronization was added when the mode was adapted to consider patient triggers in close proximity to time-triggered breaths (i.e., breaths initiated by the patient within that mandatory period). The next improvement was pressure support to breaths triggered in the spontaneous period, so we sometimes see SIMV as we described it notated as SIMV + PS. To draw these all out:[59]

Intermittent Mandatory Ventilation



SIMV (as it was known back in the day)

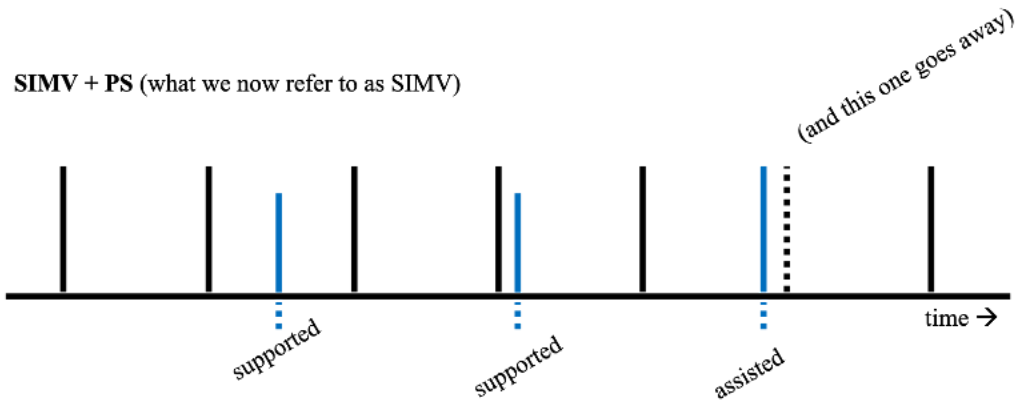


[56] Lodeserto, 2018 - This series provides an alternative explanation to this concept (i.e., how pressure support and pressure control breaths differ) and we will touch on it again in **Types of Breaths**

[57] Chatburn & friends, 2014 - And by convention both supported and assisted breaths could be labeled as assisted, it's just that pressure support breaths are a more specific type and we will discuss them that way just to remind readers about that difference

[58] Hess, 2005 - That said, the primary function of pressure support breaths is to relieve workload required by the patient and facilitate intrinsic respiratory effort, this is fundamentally different than a pressure control breath (discussed soon) in which we utilize pressure to deliver a breath regardless of patient effort; this article discusses how additional pressure support may not correlate as expected with an increase in **Tidal Volume** due to the way pressure support breaths are delivered

[59] Ghamloush & Hill, 2013 - We recognize that this confusion related to SIMV and its components is tough to follow, but navigate here for another explanation of how SIMV as we know it came to be



Another historical tidbit is that the mode was popularized as a method of weaning or getting someone transitioned from vent life to spontaneous breathing after an illness or intervention. The efficacy of SIMV for weaning has since been shown to be inferior to other methods. The result of all of this is that content on SIMV is often confounded by stuff that more accurately relates to intermittent mandatory ventilation or SIMV without pressure support and that draws conclusions from a concept (weaning) that doesn't much matter in the transport setting. Even though SIMV isn't used as often as it once was, it is important to understand that it does provide us with a valid alternative to assist control and both are legitimate choices in transport.

Now when we do put a patient on SIMV, there are some downsides to consider. First is that SIMV is less straightforward or intuitive than assist control. One component of this is that different breaths within the overall scheme may be delivered by different mechanisms and this can make it tough to manage settings when changes are warranted.[60] Another component is that supported breaths are harder to predict (in terms of volume), which may make it more difficult to meet a patient's need for more air. It can also be difficult for us to align machine effort with patient effort (i.e., SIMV is more prone to asynchrony).[61] And last thing, which may or may not be a valid reason in and of itself, SIMV is less common than assist control mode. This means that exposure to and experience with the mode is likely less and also that patient handoffs may not be as fluid as they would be if the patient were in assist control.

To summarize, SIMV is a mode that both supports patient effort to breathe via pressure support breaths and avoids breath stacking by not delivering breaths in close proximity to others. This avoids the problem of AutoPEEP that we discussed in regard to assist control. On the other hand, SIMV has been associated with ventilator asynchrony and can be harder to both conceptualize and monitor than assist control ventilation (due to the different types of breaths involved). All that said, SIMV is an appropriate mode to use in transport and provides clinicians with an alternative to assist control.

And Beyond…

Now that we know about both assist control and SIMV modes, the decision becomes which mode to use for a given patient. While many folks have their preference and we could argue one over the other all day long until we are both blue in the face, the bottom line is that either mode could work for just about any patient type. That said, this idea "that either mode could work for just about any patient type" is dependent on a thorough understanding of how a given mode works. SIMV is demonstrably more complicated than assist control and involves more variables, so that already puts it at a disadvantage. Also there is no data that SIMV is better, in

[60] We will talk further about these **Types of Breaths** in a later section, but this particular source of confusion is when we have someone in SIMV and **Volume Control**; in these cases some breaths are given by volume and others are given by pressure
[61] We will discuss this idea of ventilator synchrony in **Comfort** and then again in **Vent Graphics**

general, and there is actual data showing it is worse in certain situations.[62] Arguments in favor of SIMV, however, do exist and we'll mention two of them here. Just know that a lot of the concepts and nuances of these arguments have not yet been discussed.

First argument in favor of SIMV is related to transport specifically. In assist control, successful patient triggers always get assisted (i.e., full breaths are given).[63] If breaths are being triggered accidentally and repeatedly, maybe due to a bumpy road in an ambulance or turbulence in an aircraft, the machine gives lots of full-sized breaths back to back and that can cause problems.[64] SIMV attenuates this risk because some patient-triggered breaths will be supported (instead of assisted) and those supported breaths tend to be smaller (in terms of volume), which means a lower risk of bad things happening with too many accidentally-triggered breaths. Counterarguments: triggers can be adjusted to prevent this from happening, proper monitoring can identify this early, and this whole issue of accidentally-triggered breaths seems to be less of an issue with newer machines.

Second argument in favor of SIMV has to do with avoiding respiratory alkalosis.[65] If a patient is triggering lots of breaths in assist control, all of them will be assisted and the patient ends up moving quite a bit of volume per unit time. This can eventually create a state of respiratory alkalosis (i.e., low concentration/ content of carbon dioxide). Just as in the example above, SIMV attenuates this a bit because some of those triggered breaths would be smaller in size and therefore the degree of alkalosis is decreased somewhat. Counterarguments: respiratory alkalosis often isn't all that big of a deal and there are other ways to reduce the rate of patient-triggered breaths.[66]

Here's the general strategy we'll recommend: if we have a patient already on the vent and all is well,[67] just stick with whichever mode they are on; if we are starting from scratch or reworking the settings altogether, try what the machine defaults to (almost always this will be assist control) and then change modes if we need to down the line. Which essentially means we'll always default to assist control unless the patient is already on SIMV and we can confirm, for sure, that there are no issues.

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[62] Ghamloush & Hill, 2013 - More on that in this article that advocates for retiring SIMV as a mode of ventilation

[63] **Triggers** will eventually be discussed in depth

[64] See both **AutoPEEP** and **Asynchrony & Dyssynchrony** for more on this idea

[65] This and other acid-base issues are discussed in **Blood Gas Analysis**

[66] Reducing patient-triggered breaths can be done by addressing **Comfort** (perhaps with **Agents for Comfort and/ or Synchrony**) or fixing **Asynchrony & Dyssynchrony** patterns by manipulating settings on the vent

[67] Determining whether or not "all is well" includes many factors, including **The Three Big Things** and absence of any **Asynchrony & Dyssynchrony** patterns

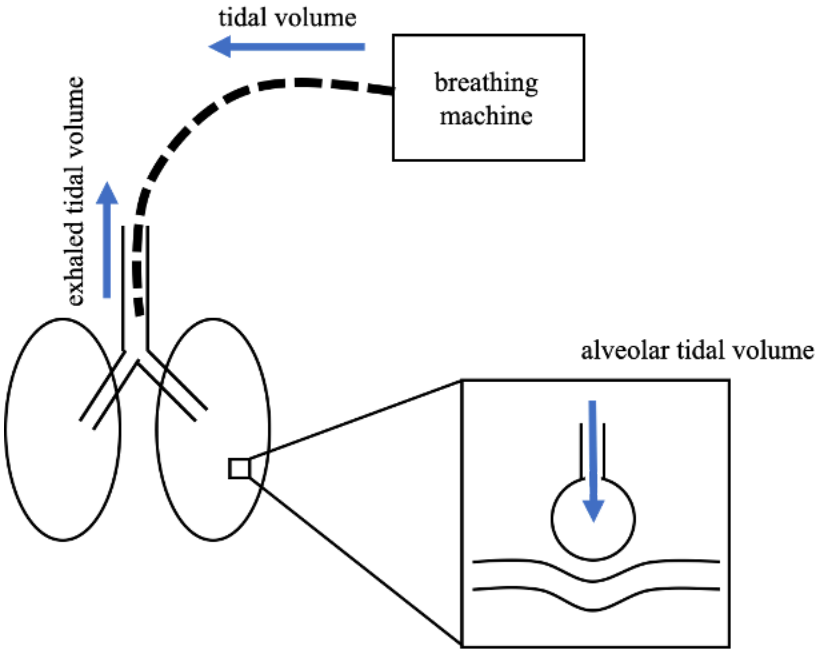
Control of Ventilation

The next decision is to choose whether we want to control volume or pressure. If we choose to control volume, airway pressure will function as the dependent variable (i.e., we won't be able to directly control it); if we choose to control pressure, volume will function as the dependent variable. There is no right or wrong answer to this dilemma, but the general trend is that we use volume control in most cases and pressure control with pediatrics or when we are especially concerned about airway pressures.[68] Not saying this is the best decision, just saying that's how it's been done.

The reason for this is twofold. First (and arguably most relevant), the machines tend to default to volume control unless we do something to intentionally get out of it. Second, volume control is a bit easier for some folks to wrap their heads around. It's a little more intuitive to think about set volumes and resultant pressures than it is the other way. But as we said above, there is no right or wrong; we can just as effectively and safely ventilate a baby in volume control as we can an adult in pressure control (even though this is contrary to what we normally do), as long as we know the underlying concepts and keep an eye on all the important things along the way!

Volume Control

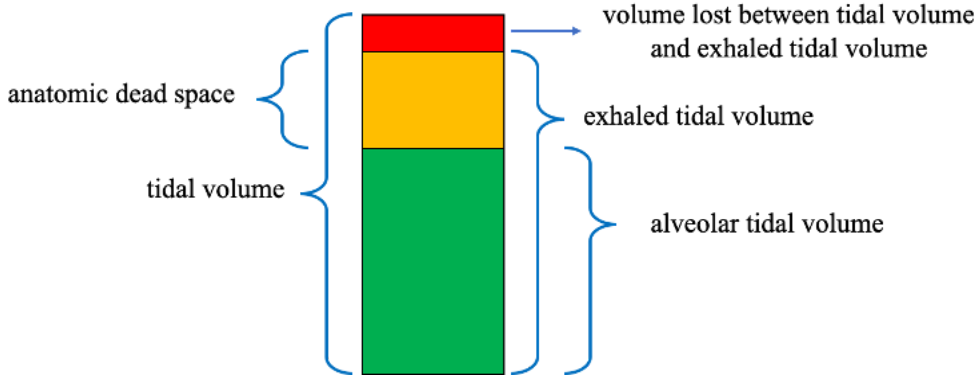
In volume control ventilation we choose how much volume we want to push down the circuit with each breath delivered.[69] This tidal volume that we put in goes to the lungs, does its thing at the alveolar level, and then gets exhaled out of the circuit. When we say tidal volume we are referring to the air going into the system from the machine; those other two concepts (alveolar tidal volume and exhaled tidal volume) vary from that value due to a number of different factors. Let's see how this looks in a graphic and then we'll hash out the details:



[68] Kneyber & friends, 2017 - Note that even the people who make the rules on pediatric ventilation don't endorse one method of control over another…

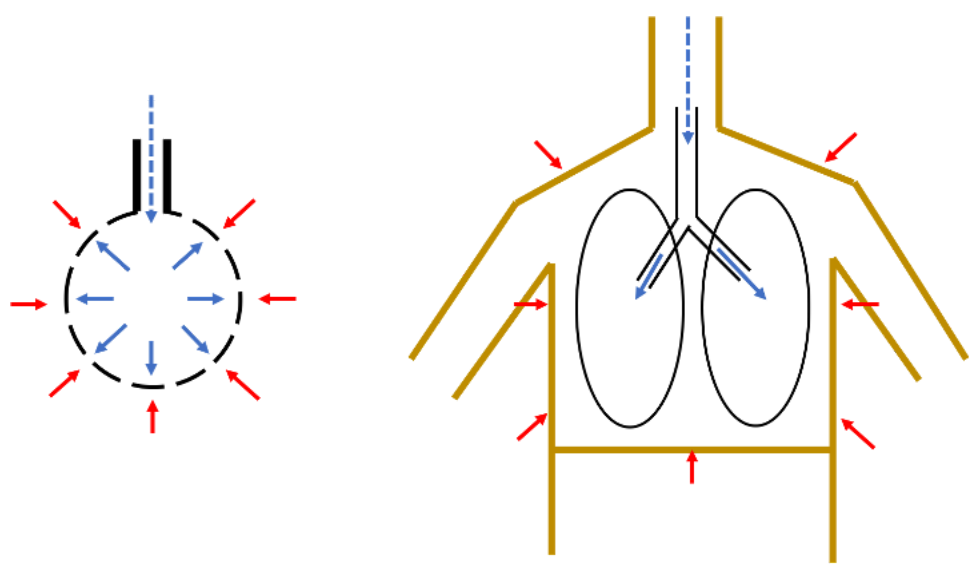
[69] To say it another way, in volume control we control **Tidal Volume** directly – a concept we mention here and then discuss again in much more detail in the near future

Exhaled tidal volume is generally about the same as tidal volume, but after some air is lost to the vent circuit and/ or to the tissues in the respiratory system. This results in the potential to overestimate volume delivered, which becomes particularly important with smaller volumes of air (i.e., pediatrics).[70] And alveolar tidal volume is exhaled tidal volume minus anatomic dead space. Recognizing the fact that not all of that alveolar tidal volume will always participate in gas exchange due to the idea of alveolar dead space, the volume of air that makes it to the alveoli is about two-thirds of what we push into the system.[71] Here's how it all looks:



While alveolar tidal volume seems a few steps removed from the tidal volume we set on the machine, volume control ventilation allows us to control alveolar tidal volume as directly as possible. The result of that, however, is an increase in pressure that is dependent on the amount of air we set and how that air moves through the respiratory system. For now we will defer a discussion of how we describe this air movement (i.e., its speed or flow), just know that pushing a preset volume in means that pressure changes happen as a result of that air movement and that certain pressure changes (i.e., too much air too fast) can cause damage to the alveoli.[72] While the alveoli do expand with added volume, at a certain point we can overinflate them. This results in what we call barotrauma or volutrauma and we for sure want to avoid those things.[73]

We will get more into all of these concepts later, but a high pressure in volume control can be due to alveoli inflated beyond their capacity, some restriction to the expansion of the chest wall, or decreased air movement through the airways:



[70] We talked about this overestimation of air delivered already in **Dead Space** and will address it again in the **Appendix**

[71] Yartsev, 2023a - This idea that alveolar tidal volume is about two-thirds of delivered tidal volume gets a bit more complicated, but we'll get back to it in **Ventilation**; for now take a look at the linked webpage for more on the idea

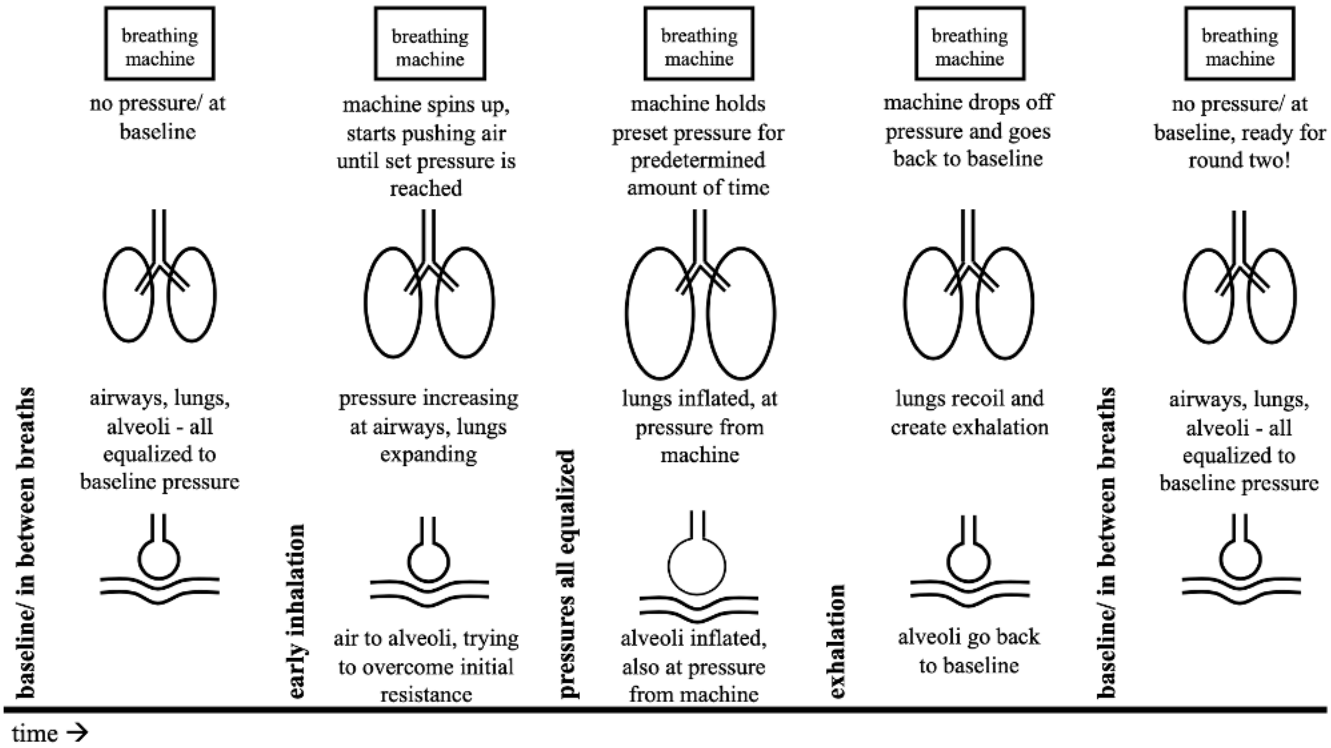
[72] Flowers & friends, 2019 - As another example of Gas Laws in action, this process is partly described by Boyle's Law (pressure change as a result of volume increase)

[73] Yartsev, 2025d - Mentioned already when these terms first came up, but look here for an in-depth discussion

The risk we face in volume control ventilation is that we can injure the alveoli or cause trauma when they are overinflated, as shown above on the left. To say this another way: there are three distinct reasons why we may see high pressures and one of those three, overdistended alveoli, is our focus of concern in this discussion. We avoid this by monitoring airway pressures and adjusting the volume input to avoid causing damage.[74] We will get to the specifics as to how we do that eventually, for now it's OK to leave it as so: in volume control ventilation we control the amount of air going into the circuit and give up control over resultant pressures. That said, we always need to monitor airway pressures during volume control ventilation in order to avoid causing damage to the alveoli. In addition, volume control lends itself to an overestimation of alveolar tidal volume if we forget to factor in dead space.

Pressure Control

In the other corner of the arena we have pressure control ventilation.[75] In this system, a breath happens as so: we have a dialed-in pressure, the machine spins up to maintain that pressure, the air all the way from machine to alveoli equalizes to this pressure for an amount of time, then the breath cycles off and we go back to baseline. Because our input here is pressure, volume becomes our dependent variable (exhaled tidal volume, to be exact and as we noted before).[76] Let's draw it out and see if we can make it a little clearer:



[74] While there are other parameters that we can adjust to avoid higher pressures (which we will get to later), it helps to simplify things this way: more volume = more pressure

[75] Meeks, 2018; Yartsev, 2022b - And we phrased it this way because there is much debate out there in vent world as to which strategy (volume or pressure) is superior; see referenced podcast and article for more information

[76] And if a machine is capable of pressure control ventilation it will surely have a mechanism for measuring exhaled tidal volume; in the previous section we noted that some machines don't give us this value, but those machines tend to do volume control ventilation only

In the fourth column, we see that recoil of the lungs (a passive exhalation) occurs when the pressure that had been keeping those lungs inflated drops off. This volume of air that gets pushed out of the circuit as the lungs fall back to baseline is our exhaled tidal volume. We then have to actively observe to make sure this volume meets the goal we have in mind for what volume this patient ought to be getting with each breath delivered. If this exhaled tidal volume is not what we want it to be, then we adjust the pressure in the system to get closer to our goal: more pressure means more volume, less pressure means less volume.

One thing worth pointing out here is that in pressure control ventilation we don't have to bother with considering that flexibility or stretch that we discussed when we talked about dead space (i.e., the compliance of the vent circuit), as the only way we have to measure volume is via exhaled tidal volume or what the patient breathes out (which is downstream of all that flexing). We do still need to consider anatomic and alveolar dead space, just as we did with volume control, but the stretch factor we introduce in our circuit is eliminated. This is a big advantage of pressure control ventilation with small patients: forgetting to factor in 10ml (arbitrary number) in an adult is no big deal, forgetting to do so for a baby with small tidal volumes is huge.[77]

Another benefit of pressure control is that we avoid the risk of overinflation or high pressures at the alveolar level. The highest pressure those alveoli will see is whatever value we program into the machine.[78] As long as we follow some basic guidelines as to what a safe pressure is, there's not much risk of harm or barotrauma. The downside is that we don't have as good of control (compared to volume control) over the amount or volume of air that we are putting into the system; instead we have to continually monitor exhaled tidal volumes and adjust to our goals.[79]

To summarize: in pressure control ventilation we control the pressure put into the system and lose direct control over resultant volumes. That said, we always need to monitor those volumes when we have a patient in pressure control in order to avoid hyper- or hypoventilation. In addition, pressure control ventilation makes it a little more difficult to manage ventilation (as opposed to oxygenation – again, concepts we will get to later on), due to the breath to breath variability in volumes.[80] The big advantage of pressure control is that we avoid the high pressures that can result from volume control.[81]

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[77] We will address the idea of how volume control can lead to an overestimation of actual volume delivered (and how pressure control can mitigate that) in the **Appendix**

[78] While it is generally true that the value dialed in for pressure control is the highest pressure the system will see as a breath is delivered, there are some exceptions that we'll chat about in **Airway Pressures in Pressure Control?**

[79] Ashworth & friends, 2018 - Refer to this article for a much more detailed discussion of how we can work towards our ventilation goals in pressure control ventilation

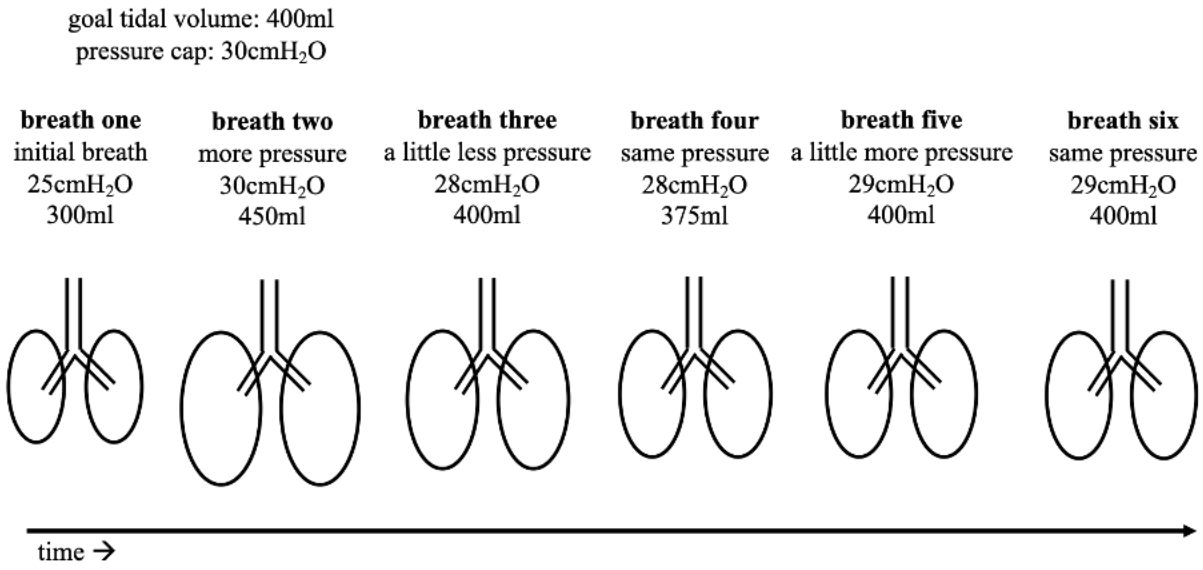
[80] And feel free to skip ahead and look at these sections: **Ventilation** and **Oxygenation**

[81] There are more advantages to pressure control ventilation (such as how pressure control breaths differ from volume control ones in regard to flow waveforms), but we'll get to that stuff later on in **Types of Breaths** and also in **Vent Graphics**

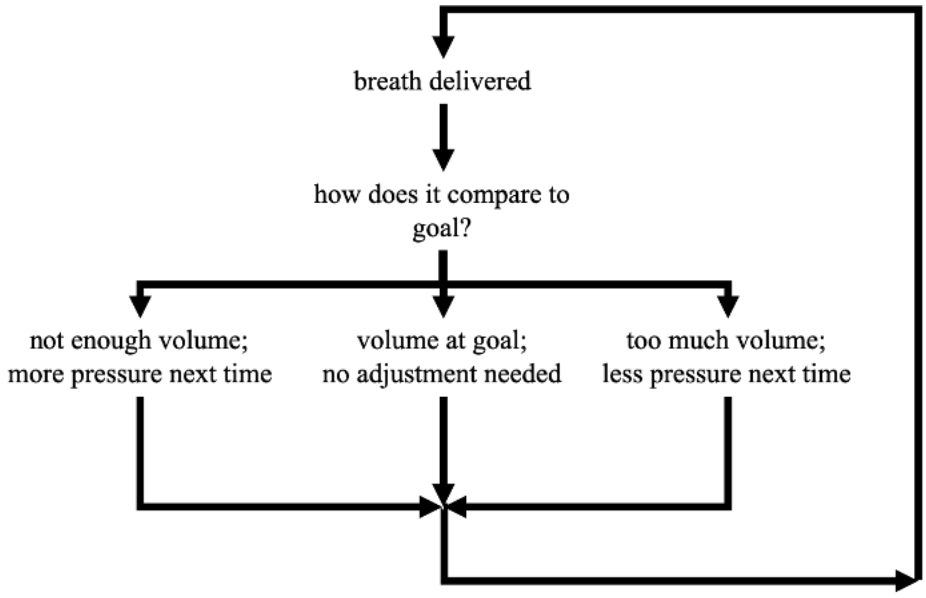
Pressure-Regulated Volume Control

Pressure-regulated volume control (PRVC) is one attempt to get at the best of both worlds when it comes to the volume versus pressure conundrum. In this type of ventilation we dial in a goal for tidal volume and put an upper limit on pressure, then the machine tries to give breaths to the goal volume using the lowest possible pressure and without exceeding the limit we have set.[82] The machine makes adjustments to how it delivers each breath by looking at previous breaths and adjusting delivery to add or take away volume working towards the preset tidal volume goal. In the event that it can't reach the goal volume without exceeding the upper pressure limit, volume is sacrificed – think of the pressure regulated part as a hard stop.

Let's visualize this over a few breaths to see what it would look like:



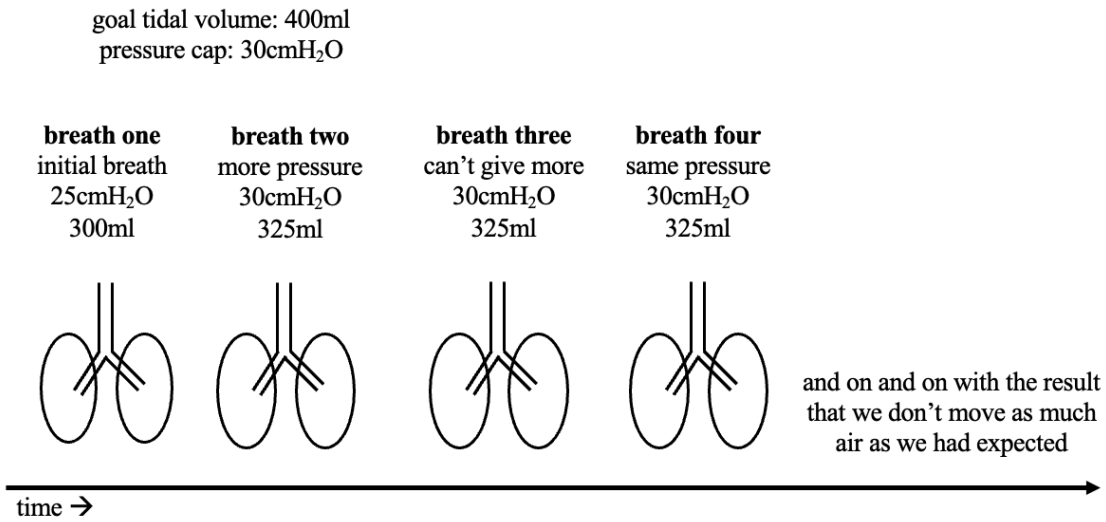
We can also think of this in an algorithmic fashion where the machine decides where each breath ends up in relation to our goal and then adjusts the subsequent breath in a cyclical manner:



[82] In the first graphic on this page and moving forward we call this pressure limit in a PRVC breath the pressure cap for lack of a better term

This mechanism of decision making one breath at a time doesn't quite describe the process accurately, but it gives the right idea. In reality, the machine looks back at the last few breaths and builds a small data set from which it decides how to deliver the next breath. So the system is more refined than our crude representation.

To flush out a few more details on this PRVC concept, let's look at another example of a few consecutive breaths. In this example, breaths fall under goal (in terms of volume). The result of this would be a drop in minute volume or air moved per unit time.[83] It's important to keep this in mind with PRVC, as we can inadvertently lose some minute volume in an effort to avoid high pressures:



A few more things about PRVC: pressure cap is a make-believe term and the machine most often uses 5cmH_2O less than the set high-pressure limit for this value.[84] There are also restrictions on how much variation occurs from one breath to the next. To say it another way, the machine won't make drastic changes in response to one or two funky breaths. Another thing: the machine has a system to get this whole process started by giving test breaths via different methods when it first gets set up.[85] Along that same idea, the machine doesn't know how much air (i.e., **Tidal Volume**) it gives with each breath until after the fact when it sees the exhaled tidal volume, that's why it can overshoot the goal. Last thing: PRVC is good when we are worried about barotrauma or giving too much pressure, but it is important to make sure we keep an eye on volumes delivered and compare it to our calculated goals.

[83] This concept of **Minute Volume** is discussed in much more detail in just a few sections
[84] And limits are discussed later when we get to **Alarms**
[85] Maher, 2019 - Short video that describes this idea of test breaths and gives a brief overview of PRVC (and it is just one video in a large series, so take a look at the rest of his content for more)

Vent Parameters Round One

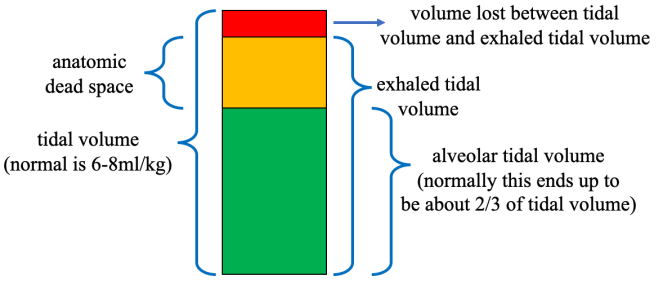
The next step on our journey is to explain the ins and outs of some of the terms we use to describe different aspects of ventilation. Some of these have been mentioned already (and a few discussed in detail), but most of the complete explanations have been left out up until this point in an effort to better organize thoughts in a linear, stepwise fashion. If it helps to go back to previous sections after this discussion, go for it. Also, keep in mind that this is not an exhaustive list of all the terms, these are just the basics and more will come later.

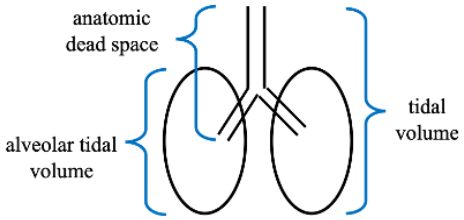
Tidal Volume

Tidal volume is the amount of air moved in a given breath. As previously discussed, it helps to break this concept up into two distinct terms: tidal volume and exhaled tidal volume.[86] Tidal volume, in this way of thinking, would be the volume of air we put into the system, while exhaled tidal volume would be the volume of air that comes out of the system. Tidal volume may be notated as TV or Vt; exhaled tidal volume is notated as VTe or Vte.

Tidal volume varies with the size of the patient and the normal range is 6-8ml/kg of **Ideal Body Weight**. Recall the discussion we already had about IBW and the idea that lung size is best correlated to height. Also recognize that 6-8ml/kg IBW is just a framework from which we start when determining our initial settings and that tidal volume can range from 4-12ml/kg IBW, depending on the specific clinical situation.[87] We will talk further on that when we get into **Specific Vent Strategies**.

And just to recap what we already discussed, add in some values, and then demonstrate it again in a slightly different way:





One more thing to mention here. We have a quantity of air in that last graphic labeled "volume lost between tidal volume and exhaled tidal volume" and this is generally an OK assumption to make (as we discussed before in **Volume Control**), but it isn't always the case because exhaled tidal volume can sometimes exceed tidal volume for various reasons. For example, there may be some breath to breath variation with the spontaneously breathing patient or (s)he may forcibly exhale after a breath is given. There is also the idea that cold air from the supply or machine end of the system will warm and expand as it enters the airways and lungs.[88] These are just a few examples, we just wanted to point out that exhaled tidal volume doesn't always have to be less than tidal volume and that reasons for this vary widely.

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[86] Tidal volume and exhaled tidal volume were first discussed back in **Volume Control**

[87] Davies & friends, 2016 - These guys offer a much more in-depth discussion about this idea of having a preset tidal volume goal for all patient populations and why we should or shouldn't deviate from that framework in our vent management

[88] Flowers & friends, 2019 - This idea of cold air expanding with an increase in temperature would be an example of Charles' Law

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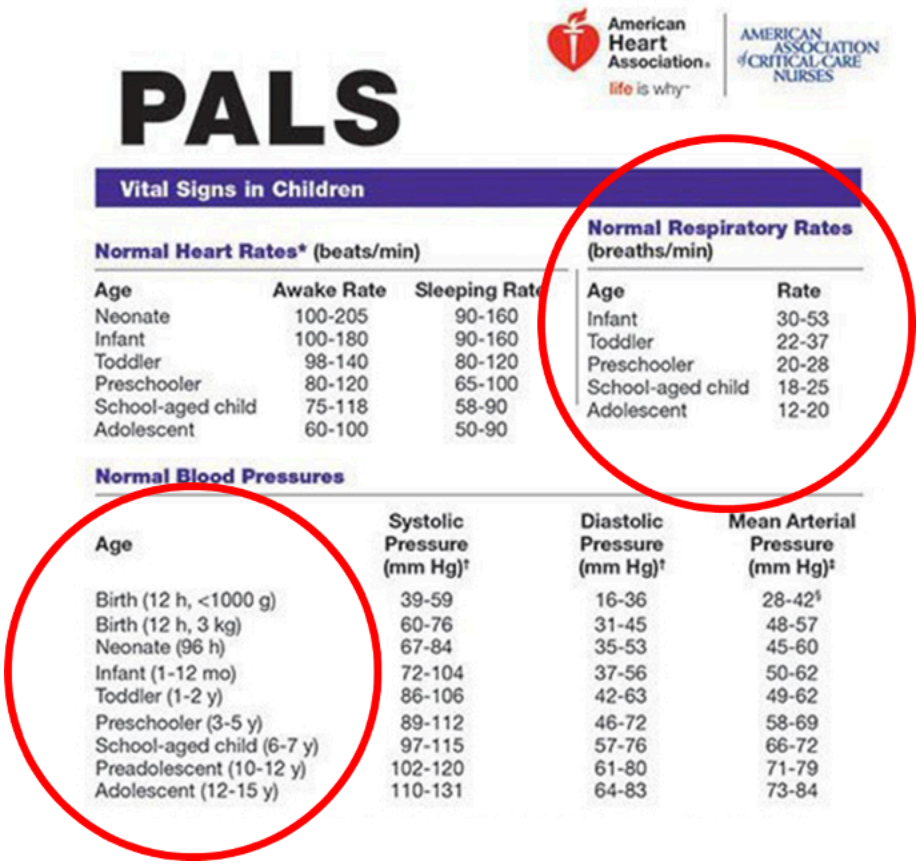
Respiratory Rate

Respiratory rate (RR) describes how many breaths are delivered and/ or taken in one minute. While rate is often considered the value we put into the machine and frequency (f) is the total number of breaths per minute after patient-triggered breaths are considered, we will use respiratory rate, or simply rate, to describe this concept as a whole moving forward. With that said, know that if we want to decrease rate and the patient is triggering breaths spontaneously, decreasing rate on the vent won't have the intended effect. No need to dwell on that idea now, we'll come back to it later on.[89]

Normal parameters vary by age, but the typical adult rate is 12-20 and pediatric rates are as outlined on our Broselow Tape[90] or by this chart from the PALS Manual:[91]



PALS

American Heart Association. life is why

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)‡
Birth (12 h, <1000 g)	39-59	16-36	28-42§
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	89-112	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

[89] This concept of an observed respiratory rate being greater than a set rate and what we can do about it is discussed in **Overbreathing**

[90] Kindig & friends, 2019 - This article gives a good overview of all the features newer Broselow Tapes have to offer, to include initial vent settings that roughly approximate the ones we discuss in this section

[91] American Heart Association, 2016 (image) - As a quick disclaimer: these normal respiratory rates as outlined in PALS are not intended to be used for determining vent settings, rather they are outlined as such to identify normal and abnormal findings in an assessment; with that said, most transport clinicians are familiar with this reference and have ready access to it, so it makes sense to build our concept of vent management from a known source rather than introduce new values and numbers with which we may not be familiar

For the detail-oriented people out there, there are some data points missing from this PALS chart. One strategy would be to guess based on available data (i.e., no listed rate for a 9-year-old, but we could assume a value that falls in between the school-aged child range and that for adolescents). Another option is to use this chart we've put together based on the existing data in the PALS chart:[92]

age description	age (years)	rate
infant	0.083 (1 month) - 1	30 - 53
toddler	1 - 2	22 - 37
preschooler	3 - 5	20 - 28
school-aged child	6 - 7	18 - 25
big kid	8 - 9	17 - 25
preadolescent	10 - 12	14 - 23
adolescent	12 - 15	12 - 20
adult	16 and up	12 - 20

Last thing: there are times that we set rate above or below what might be considered normal for the patient's age, but we'll get to those specifics when we discuss vent strategies for different situations later on.

Minute Volume

Minute volume (MV), also known as minute ventilation (VE), is the amount of air moved in and out of the lungs in one full minute. It is the product of **Tidal Volume** and **Respiratory Rate**:

$$MV = RR \times TV$$

Minute volume is the primary mechanism by which we control **Ventilation**. We will discuss how to manipulate both tidal volume and rate to address ventilation in just a bit, so don't worry about that for the moment. A normal minute volume for the adult patient is often cited at 4 – 8 liters per minute (LPM), but better practice is to use a weight-based calculation so that it applies to all patient sizes:[93]

$$MV \approx 100\text{ml/kg IBW/min}$$

Minute volume is described as roughly 100ml/kg IBW/min (i.e., ≈ and not =) because that goal is less of a hard-set requirement than a guideline by which we initiate ventilation in most cases. For the majority of patients this calculated value will be adequate, but there are times in which we ought to aim above or below for various reasons. For example, with both pediatric patients and those with **Acidosis**, we will aim higher than 100ml/kg IBW/min; with others we may tolerate a minute volume below 100ml/kg IBW/min by way of a

[92] See **Appendix** for a discussion of how this chart to outline normal respiratory rate values by age was created and see **More on Pediatric Ventilation** for details on ventilating children

[93] Weingart, 2010; Yartsev, 2023j - These cite a goal minute volume for the intubated patient as 120ml/kg/min and 70-110ml/kg/min, respectively; we've opted to go with 100ml/kg IBW/min as a starting point due to ease of calculations and simplicity; and for a discussion on weight-based minute volume goals for kiddos, see **More on Pediatric Ventilation**

concept known as permissive hypercapnia.[94] We will eventually get into the details on how we go about making that decision for different patient types, but for now we'll leave it at that.

There can be different types of minute volume, just as there were with tidal volume. Minute volume or minute ventilation typically describes what we dial into the machine, then we tag exhaled on to either term (abbreviated MVe) to describe feedback the machine gives us about what the patient breathes out. Lastly there is alveolar minute ventilation (VA) which takes out anatomic dead space from the equation. While alveolar minute volume (another way of describing alveolar minute ventilation) is an important concept to consider, we base initial goals and calculations on minute volume or exhaled minute volume and not on alveolar ventilation.[95]

Fraction of Inspired Oxygen

Fraction of inspired oxygen (FiO_2) describes the amount of oxygen in the mix of gases that we push into the patient's vent circuit when we give a breath. 100% oxygen would be an FiO_2 of 1.0; 21% oxygen or ambient air would be an FiO_2 of 0.21. Adjusting FiO_2 is often the easiest way we can address an **Oxygenation** issue, but we'll discuss fixing things in just a little while. FiO_2 is typically a parameter we dial in directly to the machine, but it can also be calculated based on how much oxygen we put into the machine and how much total air the machine puts out:[96]

FiO_2 = total amount of oxygen ÷ total amount of air
and then we can use the concept of flow to quantify these values in this equation:[97]

assume 10LPM of oxygen going in
and 60LPM of total flow

FiO_2 = [(10LPM x 100%) + (50LPM x 21%)] ÷ 60LPM
FiO_2 = 34% or 0.34

Now there is never really a need to do this sort of calculation, as the machine will allow us to bypass the math and directly provide a chosen FiO_2 as long as our oxygen source is adequate (such as one of those 50PSI adapters like we see on the wall of the ambulance or hospital). It's also not as straightforward as it seems, since flow is never delivered at a constant rate throughout the breath cycle - there's some back-end math done by the machine and the takeaway is that we just dial in a desired FiO_2 and assume that the machine makes it happen. All that said, we can bypass this mechanism by using a low-flow oxygen source (i.e., normal oxygen tubing)[98] and simply titrate oxygen flow towards a specific end goal. Doing this makes it difficult to know for sure what the FiO_2 is, but each manufacturer has different recommendations as to how we should estimate an FiO_2 based on the settings we have dialed in and the flow of oxygen into the system. The nuances of how FiO_2 impacts **Oxygenation** and is utilized clinically is discussed shortly.

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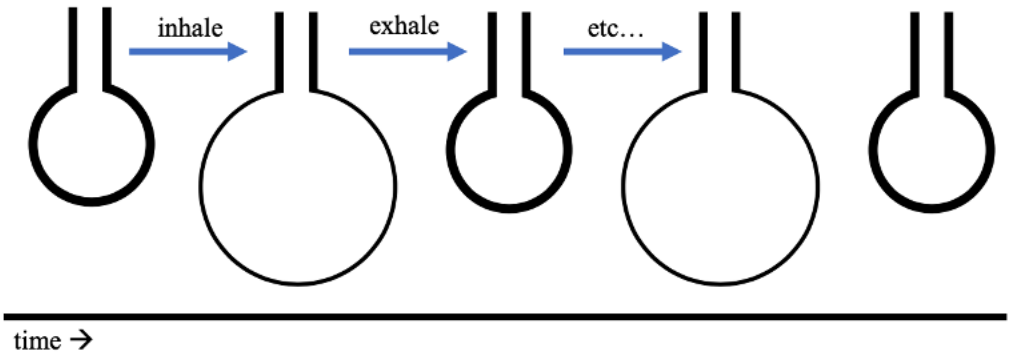
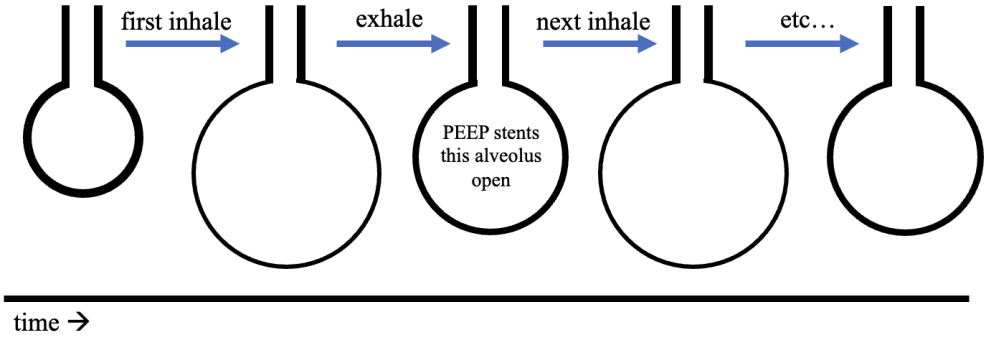
[94] Pruitt, 2007 - We cite this again later when we discuss both the **Obstruction** and **Lung Injury** strategies, but it outlines the idea of this permissive hypercapnia approach for certain patient populations
[95] We do, however, make subsequent changes to address ventilation with these alveolar volumes in mind and we will get to that in **Ventilation**
[96] Reading, 2016 - For more detail on this type of calculation to determine an FiO_2 algebraically, take a look at this article
[97] And we won't talk about flow in depth until **Types of Breaths**
[98] Hardavella & friends, 2019 - For all the details on what constitutes high-flow and low-flow systems

Positive End-Expiratory Pressure

Positive end-expiratory pressure (PEEP) describes the positive pressure that remains in the alveoli at the end of expiration. Recognizing that we basically explained a term using the words it's made up of, we'll try it another way via a few steps. During mechanical ventilation we push air into the alveoli on inspiration, then that air moves out of the alveoli on expiration. We tend to conceptualize this as a net-zero movement of air where the alveoli go from deflated to inflated and then back to deflated, as so:



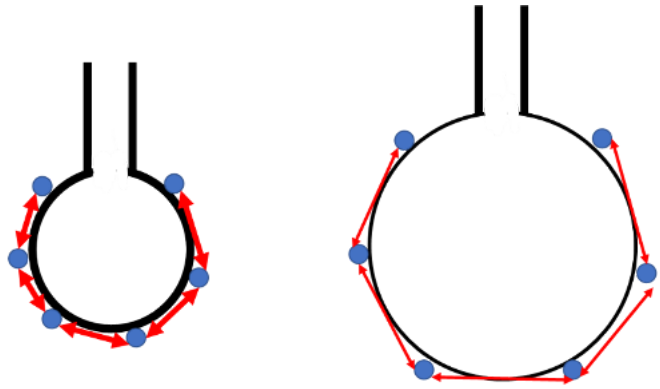
The truth is that we can put pressure into the alveoli and then maintain it throughout exhalation (in the form of PEEP). So rather than the alveolar air sac deflating all the way back to its original size, it deflates only part way:



Recall from our previous discussion of **Alveolar Surface Area** that the more inflated the alveoli are, the more they can participate in gas exchange. This is due to both more surface area and a thinner membrane across which gases must diffuse.[99] Next, add to that the idea that blood flow though the pulmonary capillary bed is continuous, it doesn't stop when inhalation stops. This means that pulmonary respiration or gas exchange across the alveolar membrane occurs throughout the respiratory cycle, both on inhale and exhale. PEEP helps facilitate this gas process on the exhalation side and then makes it easier to further maximize the effect during inhalation (i.e., a better starting point from which inhalation begins).

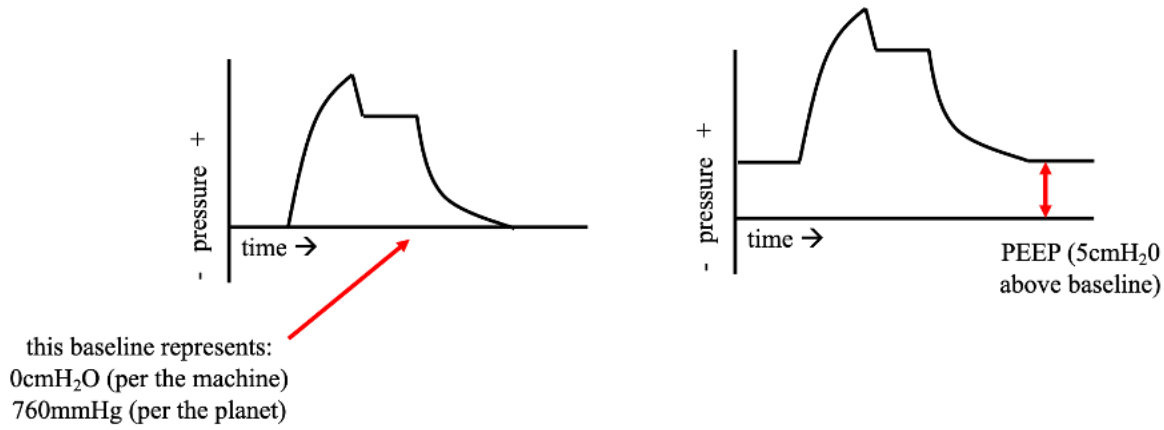
[99] Desai, 2012 - This video reviews the factors that determine the rate at which gases diffuse across this membrane; we cited it once before in **Alveolar Surface Area** and will cite it again in the section on **Oxygenation** when we return to the idea

Another idea particularly relevant to this discussion of PEEP is that the stenting or opening-up of alveoli doesn't always happen in one breath as it's been depicted in the previous drawing. Sometimes it takes time to get from that left-most, deflated stage to a recruited or opened-up stage. Part of the reason for that is that there is fluid around the surface of the alveoli that resists expansion. Think of it as molecules on the alveolar surface that are holding hands with one another; as we increase the size of the alveoli, we increase the distance between those hand-holders and make expansion easier:



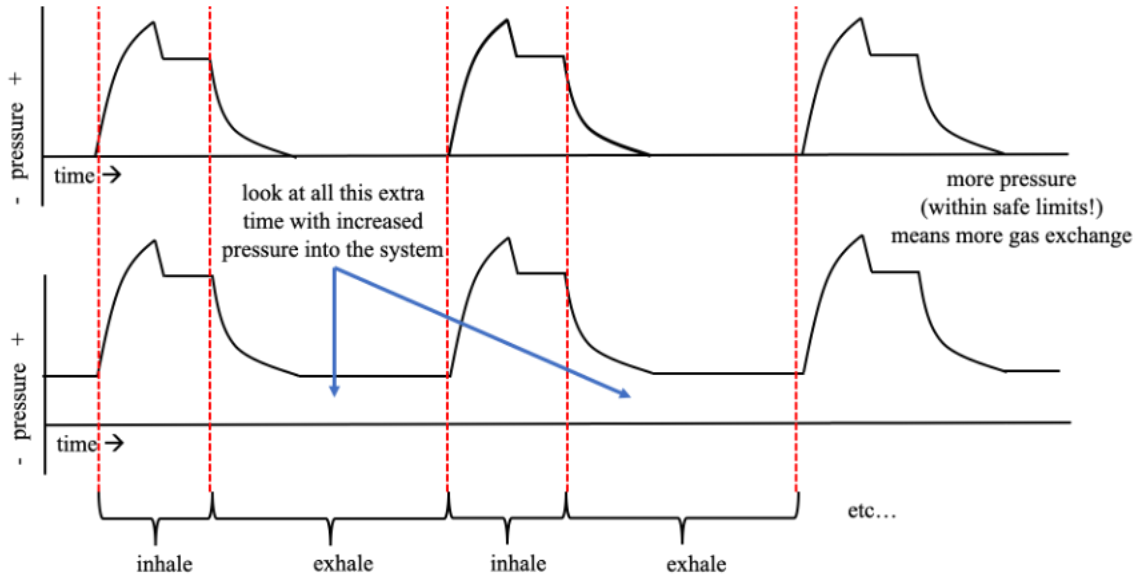
PEEP helps with this process by maintaining our progress along the way. As airway pressure increases on inhalation and the alveoli expand, PEEP essentially maintains that expansion on exhalation and prevents us from cycling back to that deflated, left-hand state in the above drawing. An added benefit of this is that it reduces stress on the alveoli. Going from deflated to inflated to deflated to inflated and on and on can damage the alveoli; PEEP decreases the difference between those two states so that less net movement is required for each inhalation. We talk about this more in the section on **Driving Pressure**, so no need for more detail at this point.

To summarize so far: PEEP is a residual pressure that we leave in the alveoli during exhalation to both maximize pulmonary respiration and maintain recruitment of alveoli.[100] So now that we have that clarified, let's look at a waveform representing pressure into the system as we deliver a breath. We've seen this image previously, but now we are going to add to it. The first breath is with no PEEP or zero PEEP or "ZEEP," the second one (right) is with 5cmH_2O worth of PEEP added in:



[100] Kallet & Branson, 2016 - While the focus of this article is on oxygen delivery (and we cite it later in **Oxygenation**), they also explain that PEEP doesn't necessarily open the alveoli as we often hear it described, rather PEEP keeps the alveoli open after inspiratory pressure changes (or **Recruitment Maneuvers**) open them up; also, to review the idea of pulmonary respiration look back to the section on **Terms to Describe Breathing**

And to visualize this same idea over time, let's think of it this way:



Now this is not to say that gas exchange is nonexistent on exhalation in the first (no PEEP) case, just that it is augmented during the second one. There are also other mechanisms by which PEEP facilitates **Oxygenation**, but those will come up shortly. The important thing to note for now is that PEEP acts to keep alveoli open during exhalation and that helps us utilize more lung volume while breathing for the patient.

Let's next take a look at the downsides of PEEP. The most relevant one to mention is that PEEP can decrease cardiac output.[101] Recall from a previous discussion that any increase in intrathoracic pressure can impede blood flow back to the heart. Because of this, normal PEEP is less than 10cmH_2O. That said, we sometimes use PEEP up to 20cmH_2O in specific cases and we will talk about those later. Other negative consequences of PEEP vary widely from things like worsening hypoxia and increased ventilation/ perfusion (V/Q) mismatch to decreased extra-thoracic organ function and decreased cerebral perfusion pressure.[102] That said, these negative effects typically manifest when the application of PEEP is taken beyond the level of therapeutic benefit. To phrase it a different way: use PEEP when needed, but don't assume it is without consequences, and be sure to utilize it judiciously.

Last point here is to mention how to choose and adjust PEEP appropriately. Note that these decisions require understanding of concepts that have not yet been discussed - we'll mention them now and then will return to them later.[103] Many ventilators default to a PEEP of 5 or 6 cmH_2O and that's totally fine for most patients. We often choose to start higher in cases of severe hypoxemia or **Lung Injury** and we may choose to start lower in marked **Hypotension** - these things are discussed later. And then we adjust PEEP to meet both **Oxygenation** and **Ventilation** goals - again, concepts that will be discussed soon. There are many different methods for optimizing PEEP for an individual patient, but the most straightforward would be to consider the effect a change in PEEP has on compliance.[104] If we change PEEP and compliance gets better, all is well and consider a subsequent change in the same direction; if compliance gets worse, go back to where we were and consider a change in the opposite direction. Again, many of the concepts mentioned in this last paragraph have not yet been discussed and this general idea of choosing a correct PEEP will come up again later.

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[101] Clinical Analysis Management, 2009; Strong, 2013 - This effect of decreased cardiac output due to PEEP isn't so much a thing with a well-hydrated patient, so we can mitigate that somewhat by giving fluids if our patient will tolerate it

[102] Coruh & Luks, 2014; Luecke & Pelosi, 2005 - Refer to these sources for detailed explanations of all of those negative consequences of PEEP

[103] This will be in both **Optimization of PEEP** and **Make a (Calculated and Informed) Plan**

[104] Edginton & friends, 2024 - This review investigates the various different methods of evaluating appropriate levels of PEEP; also see **Compliance & Resistance** for more on this concept

Let's summarize all things PEEP. PEEP is a pressure maintained at exhalation that maintains alveolar volume. This facilitates movement of gases across the alveolar membrane throughout the respiratory cycle. That said, PEEP is not without risk and therefore we must consider the potential consequences when using or increasing it. And then lastly, PEEP should eventually be optimized to an individual patient's need.

Inspiratory Time (and I:E Ratio)

The next term to consider is inspiratory time (I-time), which is the amount of time over which we deliver a breath. It varies by age as so:[105]

age description	age (years)	I-time (s)
infant	0.083 (1 month) - 1	0.3 – 0.7
toddler	1 - 2	0.4 – 0.9
preschooler	3 - 5	0.5 – 0.9
school-aged child	6 - 7	0.6 – 1.1
big kid	8 - 9	0.6 – 1.2
preadolescent	10 - 12	0.7 – 1.4
adolescent	12 - 15	0.8 – 1.7
adult	16 and up	0.8 – 1.7

One idea related to positive-pressure ventilation is that the more time we spend pushing air into the system, the more oxygen gets moved into the bloodstream. This means that more time spent on the inspiration side of the breath cycle (versus exhalation) equals better **Oxygenation**.[106] With that in mind, one way to increase time spent at inspiration would be to lengthen the inspiratory time. If we do that, however, we have to accommodate by decreasing time spent at expiration or by decreasing **Respiratory Rate**. Consider seventeen breaths over one minute:

60s ÷ 17 breaths ≈ 3.5s per breath

if in or inspiration = 1.0s,
then out or exhalation = 3.5s – 1.0s
out or exhalation = 2.5s

if we lengthen inspiratory time to 1.5s:
exhalation time = 3.5s – 1.5s
= 2.0s

[105] Chakkaparani, 2020a - And in fact, I-time may be as low as 0.2s for preterm babies
[106] This idea of utilizing inspiratory time to facilitate oxygenation is also discussed when we get to **Mean Airway Pressure**

We often represent this ratio between inspiratory time and expiratory time as an I:E ratio to describe the amount of time spent at inspiration in comparison to the amount of time spent at exhalation. A normal I:E ratio is anywhere from 1:2 – 1:3.[107] Let's build an I:E ratio for the above examples:

in the first example, we have 1.0s : 2.5s, so our I:E ratio is 1:2.5

in the second example, we lengthened our inspiratory time to 1.5s;
so we now have 1.5s : 2.0s

now we need to simplify the ratio so that one of the numbers is 1:
simply divide both sides by the smaller number: $\frac{1.5}{1.5}$: $\frac{2.0}{1.5}$
and solve for our new I:E ratio of 1:1.33

To bring it back home: we had a rate of 17 and an inspiratory time of 1.0s with a resultant I:E ratio of 1:2.5. We wanted to increase time spent at inspiration, so we changed our inspiratory time to 1.5s and ended up with an I:E ratio of 1:1.33. For now we don't have to worry about the significance of these numbers, we just need to understand the math, how we get to these numbers, and the terminology associated with them. Let's try another example, but this time we will adjust rate instead of inspiratory time:

per above: RR of 17, I-time 1.0s = I:E ratio of 1:2.5
now let's increase our rate to 20 and recalculate the I:E ratio
60s ÷ 20 breaths = 3s per breath

if in or inspiration = 1.0s, then out or exhalation = 3.0s – 1.0s
therefore out or exhalation = 2.0s

in this example, we now have 1.0s : 2.0s, so our I:E ratio is 1:2.0

now let's shorten our I-time to 0.8s and see what happens:
if in or inspiration = 0.8s, then out or exhalation = 3.0s – 0.8s
therefore out or exhalation = 2.2s

now we have 0.8s : 2.2s,
but we need to make this an I:E ratio with a value of 1:
$\frac{0.8}{0.8}$: $\frac{2.2}{0.8}$ = 1:2.75

[107] Yartsev, 2023c - To clarify this idea: a normal I:E ratio for the spontaneously breathing patient is in the neighborhood of 1:2, but often times we see something more like 1:3 with ventilated patients because we leave inspiratory time alone at a default of 1.0s – because of this convention and the facts that it is both common practice and generally well-tolerated, we've stated things as we did and will carry on with this assumption

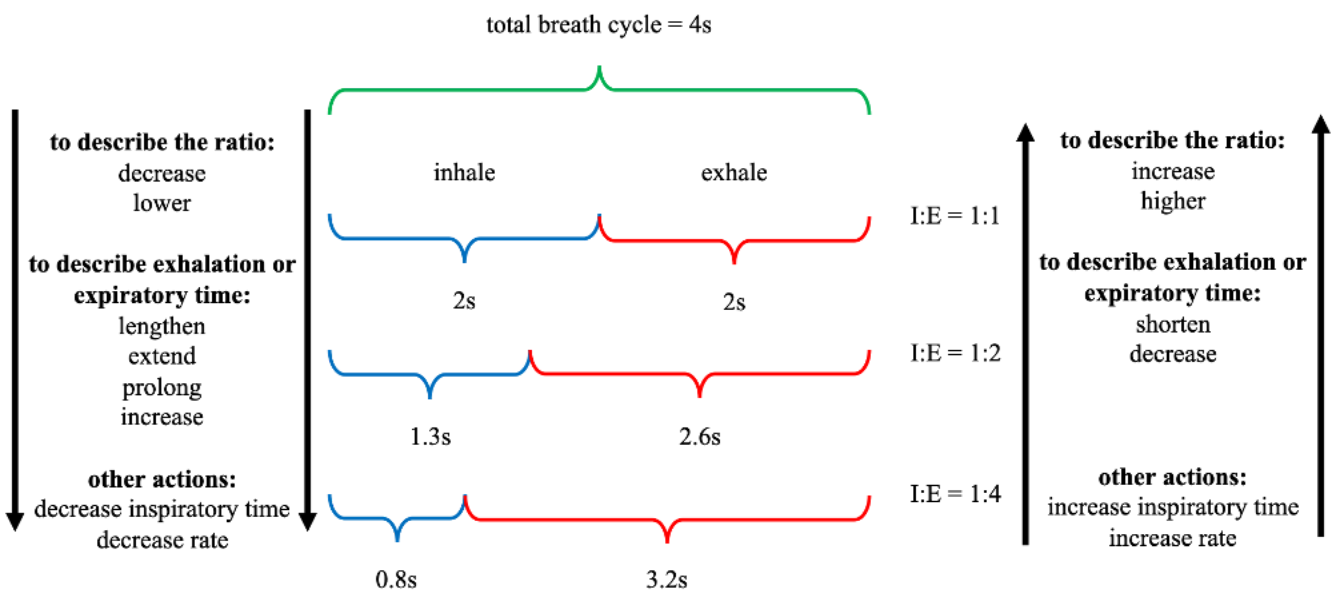
Last thing to touch on is how we describe these different I:E ratios. To make sense of this, consider the I:E ratios as fractions or decimal numbers:

$$1{:}1 = \frac{1}{1} = 1$$

$$1{:}2 = \frac{1}{2} = 0.5$$

$$1{:}4 = \frac{1}{4} = 0.25$$

In this sense, an I:E ratio of 1:2 can be decreased to 1:4 or increased to 1:1. We can decrease I:E ratio by either decreasing inspiratory time or decreasing rate. Likewise, we can increase the I:E ratio via a longer inspiratory time or higher respiratory rate. We can also describe an I:E ratio with terms such as lengthen, extend, and prolong to describe change in one direction or shorten to describe changes in the opposite direction. Just know that there isn't an accepted convention with this terminology, so to say "lengthen or extend an I:E ratio" is a bit ambiguous and could describe a change in either direction. That said, it is probably better to simply describe the expiratory time itself or to outline a specific action taken:



One final point here is that some machines allow us to select an I:E ratio directly and then inspiratory time is calculated automatically based on the chosen ratio and respiratory rate. While it is more common that inspiratory time is the independent variable and I:E ratio is calculated based on that, as we've just outlined things, both options do exist. There is also a third option in which we don't control either parameter and, instead, adjust **Flow** directly (which results in a dependent I-time and I:E ratio).[108] That said, this is not common (and maybe not even available) in transport.

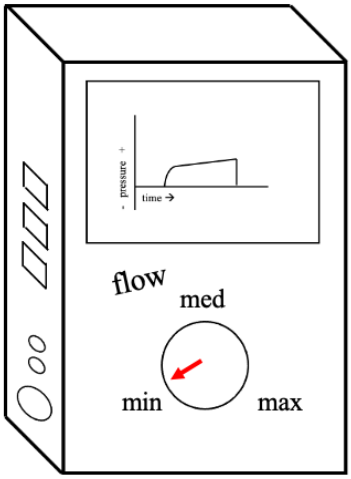
Let's summarize this all one more time and make a few generalizations: we can increase our I:E ratio by either increasing inspiratory time or increasing rate; we can decrease our I:E ratio by decreasing inspiratory time or decreasing rate. A higher I:E ratio means less time (in relation to the whole in/out cycle) spent on exhalation, a smaller or lower I:E ratio means more time for exhalation. We will talk about this later when we get to ventilator strategies, but know that some patients can benefit from a smaller I:E ratio and others can benefit from a higher I:E ratio, so it is important to know which changes affect the I:E ratio in which direction.

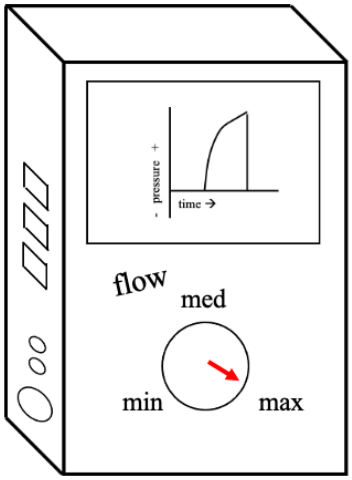
[108] This concept of flow is described in detail in the very next section

Flow

The last concept to consider in this initial batch of vent parameters is flow. Flow describes the movement of air through the system in terms of liters per minute (LPM). In transport, flow is typically calculated by the vent based on values chosen for other parameters. For example, if we are in **Volume Control** and want to give a certain amount of air over a given amount of time, flow is the result of those numbers. Or if we are in **Pressure Control** and want to maintain, say, 13cmH_2O of pressure over a breath, flow then gets adjusted by the machine automatically to maintain that.[109]

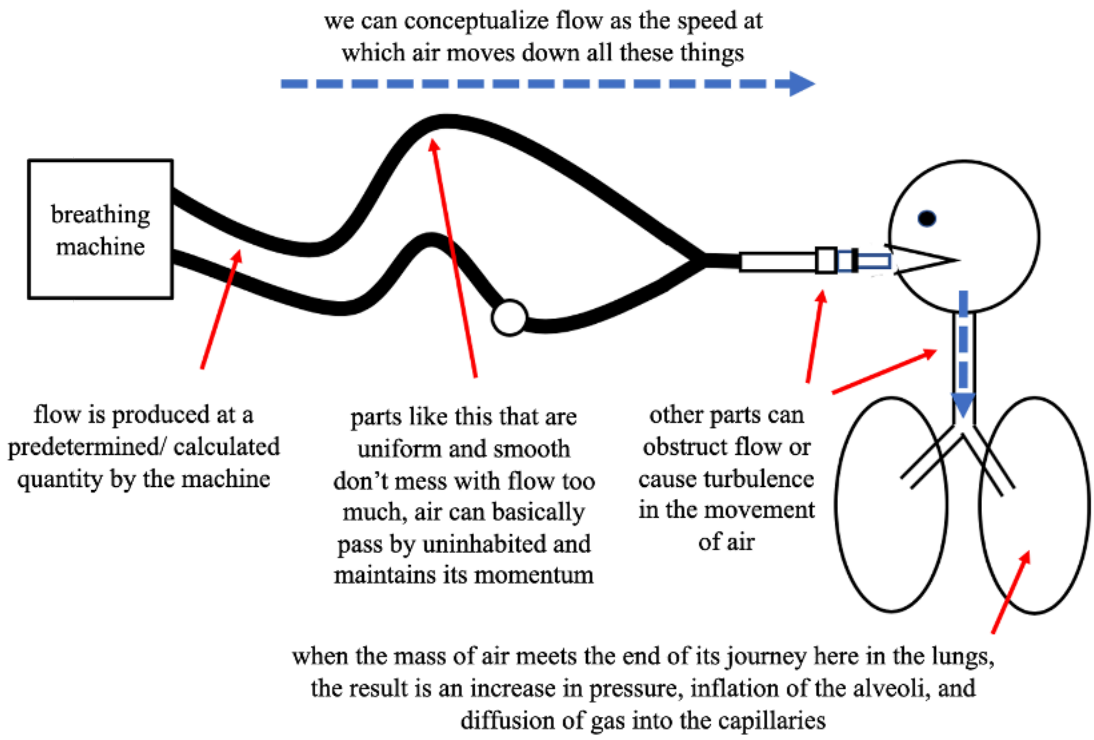
On certain vents, however, we adjust flow directly and therefore don't get to choose an inspiratory time (or I:E ratio, for that matter). We also see a difference in airway pressures. We can visualize it like this:





[109] And this adjustment is a decrease in flow over time or a decelerating flow pattern as noted in **Types of Breaths** in just a moment

Regardless of whether or not the value is determined by the machine or set by the clinician, flow is created at the machine end and dictates the speed at which air moves down the circuit into the patient:



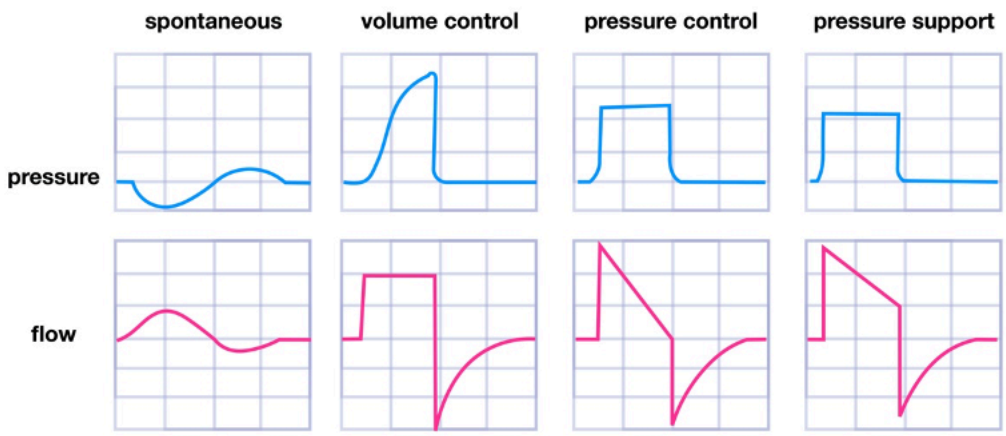
As noted in the graphic, there are things along the way which simply facilitate air movement without much of an issue and other components that disrupt flow and cause turbulence. At the terminal end, i.e., when air gets to the patient's lungs, the air then inflates the lungs by causing an increase in pressure and expansion of both the lungs and the chest cavity.

So that's the basics of flow as a general vent parameter. We'll talk a bit more about it in the next section when we differentiate different **Types of Breaths** and then we get into the specifics of analyzing **Waveforms** much later. That said, the simple concept of flow as the movement of air is something that will come up repeatedly.[110]

[110] Specifically when we talk about airway pressures in **Vent Parameters Round Two** and when we discuss **Triggers**

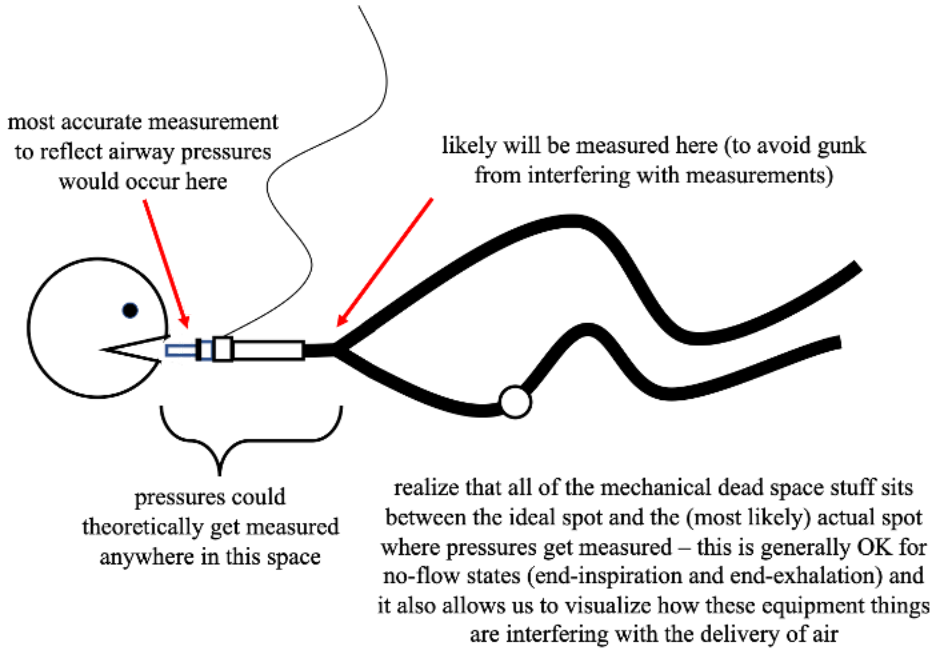
Types of Breaths

Let's take a few minutes to discuss an image we presented towards the beginning of this manual. We want to explain in a little more detail each of the following types of breaths depicted below:[111]



There are two **Waveforms** depicted for each type of breath, pressure and flow, each shown over time. We will discuss these types of **Vent Graphics** in detail later, but for now we just want to talk about the concepts in general terms. **Flow** was discussed in the previous section, so let's get on to pressure.

Pressure is measured by the machine somewhere between the endotracheal tube and the wye where the inhalation side of the circuit splits off from the exhalation side of the circuit:[112]

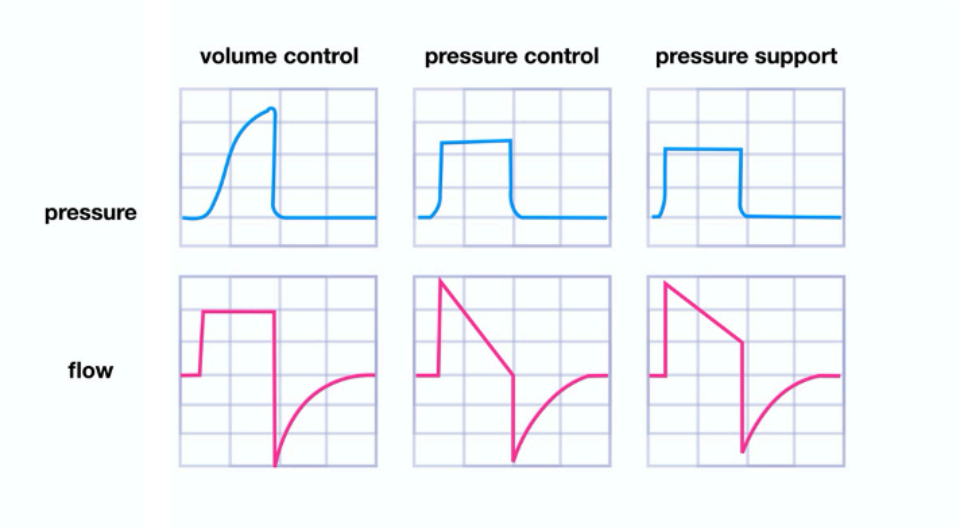


[111] We mentioned it already, but this graphic was built by Dr. Sameer of *Art by Dr. Sam* on Fiverr and *The Young Orthopod* on YouTube

[112] Hess, 2014 - We'll cite this article a few more times, it provides alternative explanations for many of the things we'll discuss along the way; specific to this section, however, it briefly describes where within the circuit pressures can be measured

Another thing to mention is that the pressures we measure don't directly describe pressures at the alveoli or terminal ends of the airway, they reflect what's going on outside of the patient's body. That said, we can manipulate the system to approximate alveolar pressures (and we will discuss how to do that later) and we assume that the value we measure correlates with average pressure at the alveoli. Pressures experienced by individual alveoli vary throughout the lung and our measurement occurs outside of the lungs themselves, but this is the best we have and therefore we base our treatment on the information available to us. The waveform that shows pressure over time gives us a visual representation of how pressure changes at the mouth side of the system as we deliver a breath. And we already talked about how pressure is measured (in terms of units), so we are good on this general idea for now.

Now that we are set on the basics of pressure (as measured in the system) and flow (as produced by the machine), let's look at a few of these waveforms again and see how we can deliver breaths in different ways:



The first thing to note is that there are three general categories: volume control breaths, pressure control breaths, and pressure support breaths.[113] In volume control, a breath is most commonly delivered via a square-waveform flow pattern in which the machine spins up to a set flow, holds it for a predetermined amount of time, then cycles off. With pressure control and pressure support breaths, however, flow is delivered via a decelerating-waveform flow pattern in which the machine starts a breath by spinning up to a max pressure and then slowly maintaining that pressure by delivering decreasing flow until the breath cycles off. To say this all another way: volume control gives constant flow for variable pressure, pressure control and pressure support give constant pressure at variable flow.

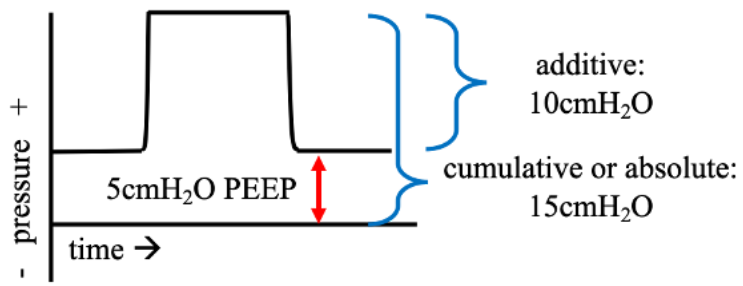
Let's follow this up with a series of sequential facts: There are some machines nowadays that can give volume control breaths via a decelerating flow pattern, but those aren't available in the transport setting.[114] That means we can lump these three types of breaths into two groups: volume/ constant flow and pressure/ decelerating flow. Unless we are in volume control and SIMV, we ventilate patients with one type of breath at a time. In very general terms: the pressure/ decelerating-waveform breaths are more comfortable for patients but take longer to deliver (i.e., may not be ideal when we need to give breaths fast or allow lots of time for exhalation).

As for the two types of pressure/ decelerating-pattern breaths (pressure control and pressure support), there are a few things to mention. First is that the pressure used to describe these breaths can either be referred

[113] And recognize that breaths delivered in **Pressure-Regulated Volume Control** fall into the **Pressure Control** category

[114] Gonzales & friends, 2012 - At least we are pretty sure the option of decelerating flow in volume control doesn't exist in any transport ventilators, but new products come up all the time; amongst many other fun things, the article explains how pressure/ decelerating-waveform breaths may be best for ARDS patients and volume/ square-waveform breaths may be best for obstruction related to COPD

to in addition to PEEP or inclusive of PEEP[115]. We describe the value as cumulative or absolute to include PEEP or additive to say it is added on top of PEEP.[116] This varies by machine, so just be aware of it:



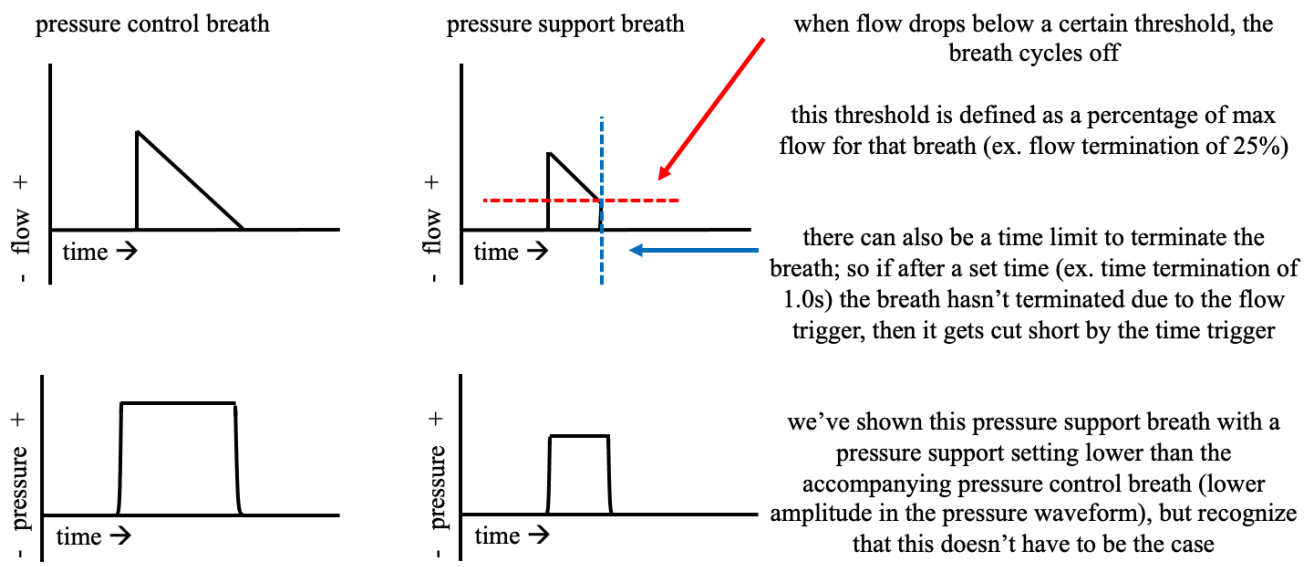
Another concept to discuss is rise time. This term describes how fast we get from zero to our set inspiratory pressure (either in pressure control or with a pressure support breath). Different machines describe and label this parameter differently, but the general idea is that a shorter rise time means the pressure gets up to what we have set more quickly.[117] This isn't something we mess around with too often in transport, but it is good to know if we are troubleshooting issues. Just keep in mind that a higher or longer rise time may mean less tidal volume if inspiratory time is not adjusted (i.e., lengthened) to accommodate that change. And then if inspiratory time changes, we may end up with less time for expiration (i.e., we will have an increased or larger I:E ratio). Again, not something we routinely mess with in invasive ventilation, but it is good to know.

[115] And as a reminder, PEEP is **Positive End-Expiratory Pressure** and we discussed it in a previous section

[116] Ashworth & friends, 2018, Bauer, 2016a - The first mentions this idea of describing pressure control or pressure support relative to PEEP in the context of pressure control ventilation; the second reviews this idea as it relates to non-invasive positive-pressure ventilation (which we don't get into here in this manual)

[117] Yartsev, 2023c - For a more detailed discussion of rise time and how things differ between machines, navigate here

The next thing to mention here is how pressure control and pressure support breaths differ. While both are given via a decelerating-flow pattern, the mechanism by which the breath cycles off changes things. A pressure control breath is designed to deliver a full breath even with no patient effort, whereas a pressure support breath is designed to simply relieve some effort of breathing on the front end of a breath. Because of this difference, a comparable titration of pressure (i.e., a change of 5cmH_2O for both pressure control and pressure support) may result in different changes of volume in the very same patient. Now the mechanism by which this works is known as termination, the parameter by which the machine decides to stop supporting a breath and begin exhalation:[118]

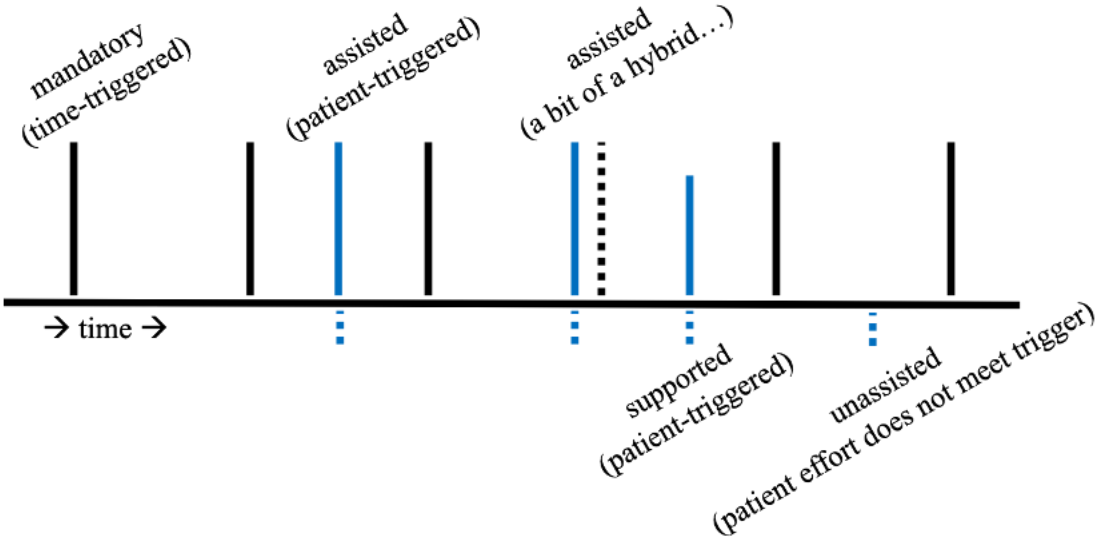


So to get more volume in a pressure support breath (represented by the area under the flow-time waveform), we either need more patient effort (i.e., don't take away a patient's respiratory drive with too much sedation) or we need to maximize our termination triggers (i.e., lower value for flow termination, longer time termination).[119] We don't typically get that far into the weeds with invasive ventilation and pressure support, but we will often see this idea discussed in terms of non-invasive ventilation (which, again, we don't cover in this manual).

[118] To expand on this, the term cycle refers to how the machine decides to stop giving a breath in a general sense, termination as we've drawn it out is specific to pressure support breaths

[119] The primary mechanism for terminating a pressure support breath will be the flow termination and it may help to think of the time termination as a backup in the event that the breath doesn't end via the flow termination mechanism

Last bit of this section: let's review different types of breaths as they relate to time-triggered, patient-triggered, assisted, supported, and unassisted (which is slightly different than they were described in that first image in the section). We touched on these as we moved through the different modes, but we'll just clarify a few things and show how they vary from one to another starting with a graphic:



Mandatory or time-triggered breaths are the ones that we deliver via our set rate on the vent and to a specific goal, whether that be volume or pressure. Assisted breaths are triggered by patient effort and then the machine delivers a full breath to match the same goal as for the machine-delivered ones.[120] Moving right, supported breaths are also patient-triggered, but get delivered via pressure support and not to a set goal. Supported breaths are often smaller than mandatory or assisted ones (in terms of volume), that's why they have been shown with a shorter blue line.[121] And lastly is spontaneous effort that doesn't get supported or assisted - these efforts get ignored by the machine and function solely via patient effort.

To take this discussion one step further, let's consider which types of breaths apply to which types of ventilation. In assist control we have time-triggered or mandatory breaths and assisted breaths. In SIMV we have time-triggered or mandatory breaths, assisted breaths (when a trigger is sensed within the mandatory period), and supported breaths (when a trigger is sensed in the spontaneous period). In neither mode do we see spontaneous effort that meets the trigger threshold and does not get assisted in some way. While there may be spontaneous effort that doesn't meet the trigger (and this theoretically could contribute some to minute volume), all noteworthy patient effort (defined by meeting whatever trigger threshold we have set) will get facilitated by the machine in some way in either mode.[122]

[120] Chatburn & friends, 2014 - For more on that hybrid situation, take a look here; these guys would identify it as a machine-triggered breath, but since we started our discussion with a differentiation between time- and patient-triggers we will just refer folks to their article – the labeling doesn't change things

[121] But again, this doesn't necessarily have to be the case and we can, in fact, work to have pressure support breaths equal in volume to those mandatory or time-triggered ones; see section on **Synchronized Intermittent Mandatory Ventilation** for more on this

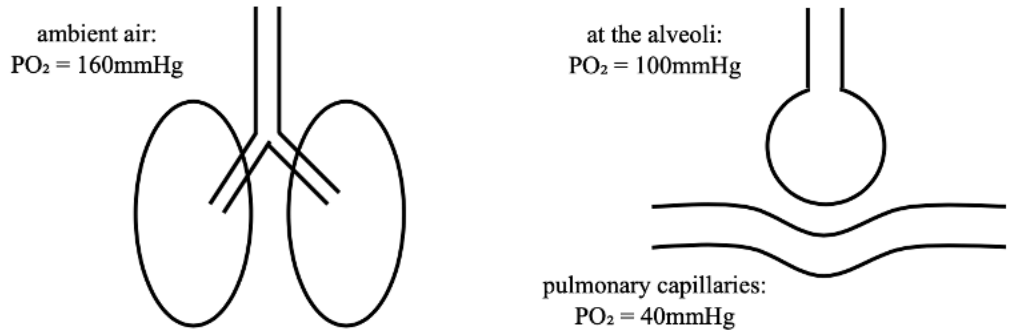
[122] And we realize that we've talked a lot about **Triggers** here, but the details on that have been deferred until later on

Three Big Things

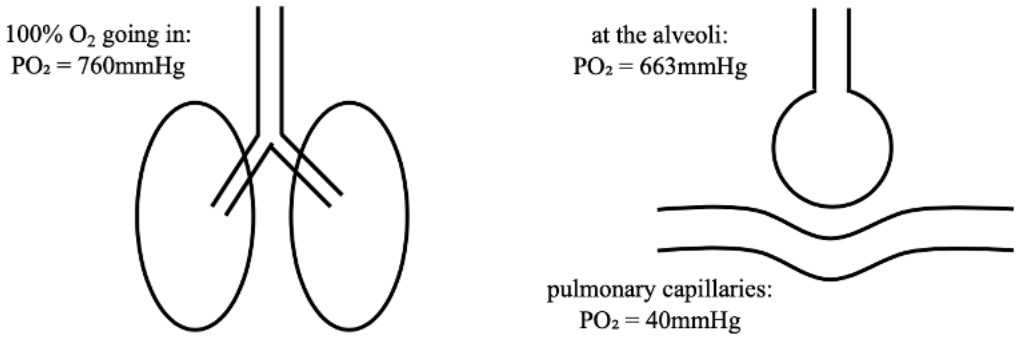
There are three very important things that need to be monitored and addressed for all ventilated patients, hands down and no matter what. The order in which we discuss them here is totally arbitrary as they all hold equal weight and are interrelated. The ideas that follow are in general terms and not specific to certain pathologies or patient types. All those details will come later. The three big things are **Oxygenation**, **Ventilation**, and **Comfort**.

Oxygenation

It may have come up once or twice before, but oxygen is pretty important stuff. Oxygen gets to tissues via a few steps, some of those we can affect directly with the ventilator. Let's start with a version of a graphic we used earlier that shows partial pressures at a few steps along the way in the spontaneously breathing patient:[123]



Gases diffuse from areas of high concentration to areas of lower concentration. In this baseline example, oxygen will move from the ambient air to the alveoli, then into the pulmonary capillaries. The first way that we can speed this process up is by changing the partial pressure of oxygen at the start of the system. Instead of 21% of the gas mix or 160mmHg of oxygen, we can titrate that all the way up to 100% (**Fraction of Inspired Oxygen** of 1.0) or 760mmHg. This will increase the rate at which oxygen diffuses to the alveoli, resulting in a higher partial pressure of oxygen downstream and, subsequently, faster diffusion into the bloodstream:[124]



[123] Bauer, 2016c - Take a look at this short article for a review of Henry's Law and the concept of PO_2 at the pulmonary capillaries, offers a preview of how we can improve oxygenation in the ventilated patient (the steps of which we will lay out in this section)
[124] This math is discussed in the **Appendix**

Let's recap this bit and do some math: PO_2 at the alveoli on ambient air is 100mmHg, PO_2 at 100% FiO_2 is 663mmHg. To quantify the result of this difference let's apply Fick's Law:[125]

$$\dot{V} = \frac{(P_1 - P_2)\ x\ area\ x\ D}{thickness}$$

$\dot{V}$ = rate of gas diffusion across a membrane (i.e., at the alveolar membrane)
P_1 = ingoing pressure (i.e., to the alveoli)
P_2 = pressure at other side (i.e., in the blood)
area = self-explanatory…
D = diffusion constant for a particular gas (oxygen in this case)
thickness = also self-explanatory…

if $\frac{area\ x\ D}{thickness}$ is constant and we call it k,
we end up with the following:

$$\dot{V} = (P_1 - P_2)k$$

and let's add in some numbers for the ambient air and 100% FiO_2 situations:

$$\dot{V}_{ambient\ air} = (100 - 40)k$$
$$= 60k$$

$$\dot{V}_{100\%\ oxygen} = (663 - 40)k$$
$$= 623k$$

That means that oxygen diffusion occurs about ten times faster at 100% (or an FiO_2 of 1.0) than at room air. Which is both nuts and a clinically significant thing to be aware of. The takeaway here is that whenever we need to increase the diffusion of gas across the alveolar membrane, FiO_2 is a heck of a way to get that done. The holdup is when other factors in the equation (area and thickness) are also issues or if the problem is with oxygen transport after the point at which it diffuses into the blood. In those cases we may need to augment this strategy with other techniques, as we will discuss real soon.[126]

Another thing to mention here is that oxygen can cause damage when given in excess of physiologic need for a sustained amount of time. Now the timeline at which the stuff can occur is likely longer than our transport, but that doesn't mean we need to get reckless and ventilate everyone with an FiO_2 of 100%. We'll talk in just a moment about how we evaluate oxygenation and the idea is to make sure a need is met while being conscious that all interventions, even something as seemingly benign as oxygen, have consequences.[127]

To expand on this idea just a bit before we move on, one specific argument against a high FiO_2 is the idea of absorption atelectasis – the closing of alveoli related to nitrogen washout and the fact that oxygen quickly diffuses into the bloodstream leaving less gas in the alveoli.[128] While the clinical impact of this sequence of events is up for debate and may not actually make a difference, it is something that comes up in the discussion of ventilated patients.[129] And if we do give credence to the idea, ways to mitigate this effect would

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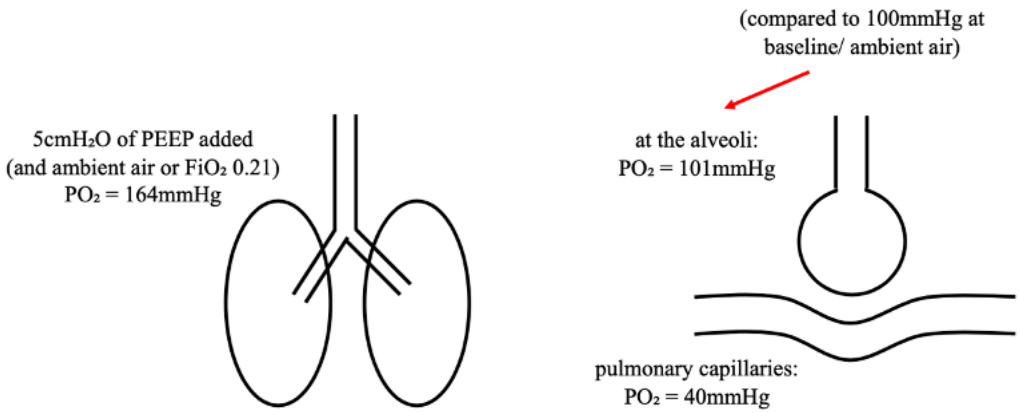
[125] Desai, 2012 - This video, courtesy of Khan Academy, outlines all of these factors with an animated depiction of Fick's Law
[126] Murphy, 2017b; Macintyre, 2014 - And to review the different types of hypoxia, take a look at this video (lots of detail, reviews the four types as we often label them in critical care transport) and that article (different system of considering the various causes, but equally informative)
[127] Kallet & Branson, 2016 - This article looks at both why it may make sense to limit oxygenation and how the negative consequences of oxygen may be exaggerated; we also have a video on this concept - *Oxygen Haterz*
[128] Dunphy, 2012 - Short video that explains both the mechanism of absorption atelectasis and how patient effort can mitigate the effect
[129] Yartsev, 2023h - This article both sheds some doubt on the idea of absorption atelectasis and describes many of the other mechanisms by which oxygen can adversely affect our patients

be maintaining a patient's spontaneous effort to breath (discussed shortly in **Comfort**) and performing **Recruitment Maneuvers** (discussed much later).[130]

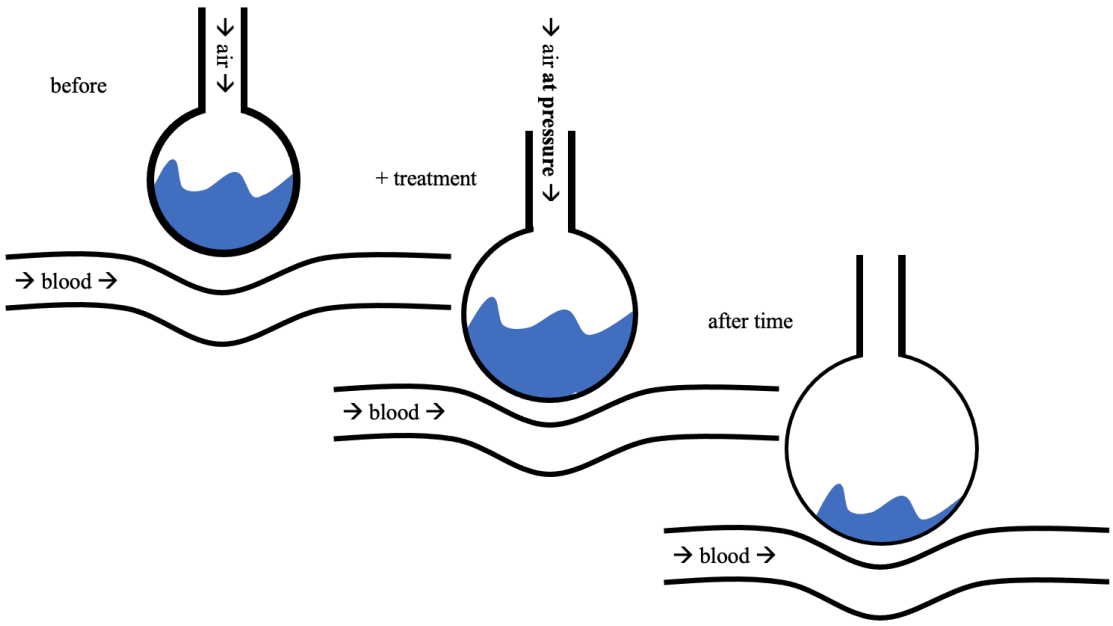
Increasing FiO_2 is one way to get more oxygen into the bloodstream. While this isn't the fix for all types of hypoxia and there are some potential negatives, we generally start here when looking to address oxygenation issues. The next way we can increase oxygenation is via **Positive End-Expiratory Pressure**. Now PEEP doesn't quite work by the same mechanism, as the addition of PEEP doesn't much change the partial pressure situation as we saw with an increase in FiO_2:[131]



[130] Hartland & friends, 2015; Radermacher & friends, 2017 - The first article outlines an argument for the use of recruitment maneuvers in certain patients (which seems reasonable to extrapolate to some of the patients we see in the transport setting); the second specifically addresses this idea that recruitment maneuvers are of benefit with absorption atelectasis

[131] Again, all of these calculations live in the **Appendix**

Instead, PEEP facilitates oxygenation primarily by increasing and thinning out the alveolar surface throughout the respiratory cycle. We discussed this concept way back in the section on **Alveolar Surface Area** and again just a moment ago in the section on **Positive End-Expiratory Pressure**, so no need to redo all of that here. One more mechanism by which PEEP helps oxygenation is that it facilitates displacement of fluid that accumulates there. While the mechanism is a bit complicated, it can help to think of it this way:[132]



As a point of clarification on this idea, fluid in the alveoli doesn't necessarily impede gas exchange as much as we might think. As we mentioned before, carbon dioxide moves fairly efficiently through liquids.[133] Oxygenation, on the other hand, can be readily maintained by increasing PO_2 at the alveoli via both FiO_2 and PEEP as we just pointed out.[134] Edema in the alveoli (pulmonary edema) impairs oxygenation in roundabout ways. We'll mention these later, but one of the primary mechanisms of this is that edema makes alveoli more susceptible to damage in positive-pressure ventilation.[135] It also makes it harder for us to get air into the alveoli and increases work of breathing.[136] While fluid in the lungs does, in fact, impact the processes of oxygenation and ventilation, it's not exactly a straightforward diffusion problem.

But to get back on track with our discussion: assuming **Ventilation** and **Comfort** are adequate (see next sections), initial steps to fix oxygenation are increasing FiO_2 and then adding PEEP. While it's totally OK to use a stepwise approach that titrates both FiO_2 and PEEP in line with one another, recognize that FiO_2 is our most direct fix for improving partial pressure of oxygen at the alveoli and has few consequences in the acute

[132] Taenaka & Matthay, 2025; Garcia-Delgado & friends, 2010 - The idea that PEEP pushes fluid out of the lungs may not be as straightforward as depicted, but it is true that there is a correlation between applied PEEP and fluid clearance, it is likely related to improved oxygenation (as noted) and better lymphatic drainage; the first article discusses how hypoxia and hypercapnia (both mitigated by PEEP) impair alveolar fluid clearance; the second describes an experiment in pigs that demonstrates a difference in fluid clearance with and without PEEP

[133] Speller, 2018 - We first cited this article in **The Normal Breathing Process** when we talked about how both oxygen and carbon dioxide diffuse across the alveolar-capillary membrane

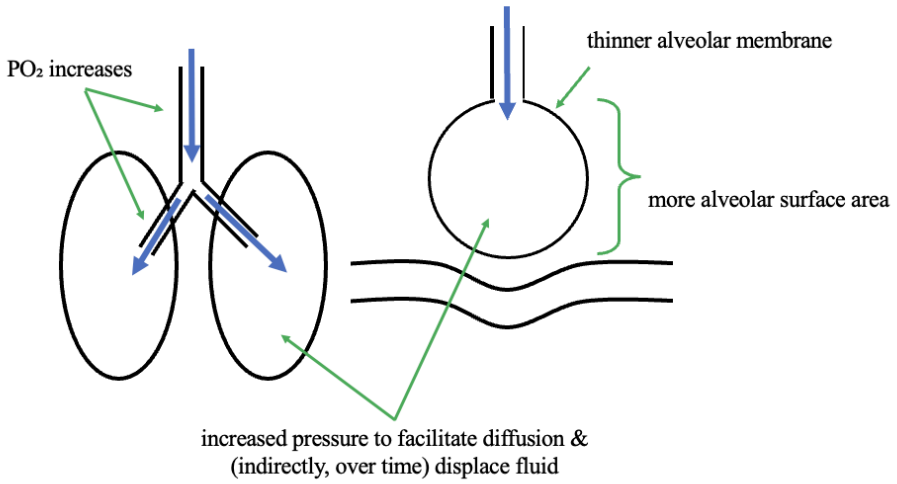
[134] Radermacher & friends, 2017 - This article discusses the management of ARDS (which we will get to later) and mentions this idea that the movement of gases through liquid in the alveoli can be overcome relatively easily

[135] Perlman & friends, 2010 - Damage to the alveoli, whether from edema or any other cause, can lead to a cascade of effects that further impact both oxygenation and ventilation; this paper focuses on the specifics of how individual alveoli are affected by edema in neighboring alveoli

[136] Kuhn & friends, 2016 - Another way to say this is that edema decreases compliance (see **Compliance & Resistance** for more)

(i.e., short term) setting.[137] PEEP, on the other hand, is especially helpful in facilitating gas exchange across the alveolar membrane and driving fluid out of the lungs, but may decrease cardiac output by way of a drop in preload to the heart (especially if our patient is down on fluids).[138] Lastly, both of these techniques (FiO_2 and PEEP) improve oxygenation throughout the respiratory cycle.

The next point to make is that all of the benefits of FiO_2 and PEEP (in the context of oxygenation) are further maximized during inspiration:



This means that more time spent at inspiration further maximizes oxygenation, therefore strategy number three to improve oxygenation is to increase **Inspiratory Time**. And changing inspiratory time consequently changes our I:E ratio. More specifically, increasing inspiratory time increases our I:E ratio (and shortens the expiratory time). For example, if we have an I:E ratio of 1:2 and then increase inspiratory time we might get an I:E ratio of 1:1. And then if we increase it enough, it will eventually become longer than exhalation and we end up with an inverted I:E ratio that might be written as 2:1. The primary drawback of a long inspiratory time (and of an inverted I:E ratio) is that it can be uncomfortable for our patients and we will need to get aggressive to maintain patient **Comfort**. An inverted I:E ratio may also make it tough for the patient to exhale fully, predisposing us to **AutoPEEP**.

Summary up to this point is that there are three ways to improve oxygenation by adjusting settings on the vent: increase FiO_2, add PEEP, and extend inspiratory time. Now why do we not just fill the lungs up with 100% oxygen and keep them inflated – we'd have a forever-long maximum diffusion of oxygen into the bloodstream, right?[139] There are two reasons for this. One is that we don't want to affect hemodynamics indefinitely (as discussed above and previously). Two is that it isn't all about oxygen. We also have to consider its partner in crime, carbon dioxide, which doesn't diffuse so well in gas (as compared to oxygen) because it is a bigger, heavier molecule.[140] The movement of carbon dioxide, therefore, is partially dependent on the movement of the body of air in which it hangs out. And that leads us into our next section on **Ventilation**, but a few more things to cover before we get there.

Other things we can do to improve oxygenation include sitting our patient upright or elevating the head of bed,[141] ensuring adequate perfusion, utilizing more lung volume via **Recruitment Maneuvers**, and considering **Mean Airway Pressure**.[142] We won't get into the details of all of these things here, as the focus for now is on the basics.

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[137] We'll touch a bit more on this subject of titrating FiO_2 and PEEP together in the section on **Lung Injury**

[138] These negative consequences of positive-pressure ventilation were discussed in both **How is Positive-Pressure Ventilation Different?** and **Positive End-Expiratory Pressure**

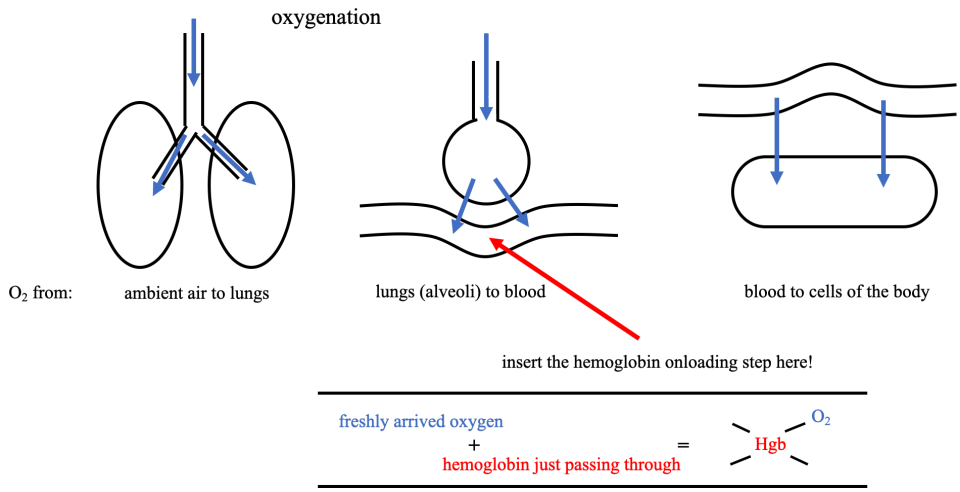
[139] **Airway Pressure Release Ventilation** (APRV) is about as close as we can get to this idea, see discuss of that mode for details

[140] Flowers & friends, 2019 - This difference in how quickly oxygen and carbon dioxide move is explained by Graham's Law

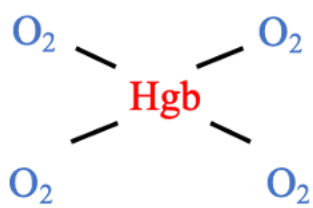
[141] Spooner & friends, 2014 - While this article mainly focuses on lung volumes (which is a step or two removed from oxygenation), it goes into detail on the physiologic effects of elevating the ventilated patient's head

[142] While we could also make the argument that going up on respiratory rate increases the amount of time spent on inspiration, doing so also impacts ventilation (next section) so we generally don't consider rate one of the variables by which we control oxygenation

One more thing to consider is how we measure oxygenation. The standard tool in the field is pulse oximetry or SpO_2. Pulse oximetry uses infrared to see to what extent our hemoglobin is saturated with oxygen (or oxygen-like things, but we won't worry about the tricky parts here).[143] The process goes like so: oxygen gets to the alveoli, it crosses into the bloodstream via diffusion gradients, then once in the bloodstream it gets picked up by hemoglobin for a ride down the blood vessel:



So we have a hemoglobin with four sites, one of which is occupied by an oxygen molecule and the resultant hypothetical SpO_2 here is 25% (1 of 4 sites filled). Fill all four sites up and we are 100% saturated as so:



Drawing it out this way is a bit of a simplification, but it does help us to understand what it is that pulse oximetry is looking at. Do note that hemoglobin doesn't cruise freely through the vessels, it comes attached to red blood cells (lots and lots of hemoglobin per each red blood cell), but the four sites per hemoglobin is a fair description. Also consider that we measure this saturation peripherally (hence the p in SpO_2). This means that if blood isn't getting to the periphery where we have our probe attached, numbers may not be accurate. And lastly, recall that as oxygen binds to that first site on the hemoglobin the physical shape of the hemoglobin molecule changes to attract subsequent oxygen molecules to the remaining vacant sites. This is why we aim for higher pulse oximetry values over 93% - once we get those hemoglobin molecules mostly filled up, it makes it way easier to fill up the remaining ones.

To carry on with this idea: when we get to 100% saturation, all further oxygen we put into the system will remain as dissolved oxygen in the blood. This has the potential to cause damage (as we've discussed before), so we tend to titrate pulse oximetry to an upper limit of 99%.[144] We don't normally take our assessment of oxygenation much further than this in transport, but there are some programs that have the ability to measure blood gases, so let's just touch on that for a moment.[145] Partial pressure of oxygen (PO_2) allows us to see how

[143] Silverston, 2016 - Short article that describes both the technology and the limitations of pulse oximetry
[144] And for more on this concept, take a look at our YouTube video *Oxygen Haterz* on the actual risks involved with oxygen
[145] Also see the section **Blood Gas Analysis** for further detail

much oxygen is dissolved in the bloodstream; normally we consider PaO_2 or arterial partial pressure in these clinical situations. If we have an SpO_2 of 100% and a normal PaO_2, then our potential for causing damage is less than if we had a saturation of 100% and a markedly elevated PaO_2.

There are, however, many things that impact this relationship. Different factors can change hemoglobin's affinity for oxygen (and carbon dioxide) and we can better understand values for SpO_2 and its relationship to PaO_2 by considering this effect. For example, we may see a low SpO_2 paired with a normal PaO_2 to indicate that hemoglobin isn't holding on to oxygen as well as normal. Or the classic example in transport: increased **Ventilation** with overzealous bag-valve mask technique resulting in an SpO_2 that looks OK, but is masking a hypoxemic state.[146] This gets a bit beyond the scope of our discussion, but we'll return back to this idea later on when we talk about **Acidosis** to give some concrete examples and clinical application.[147] Just keep in mind that PaO_2 provides a snapshot in time, while pulse oximetry provides a continuous stream of information.[148]

One very last thing about this and then we'll get on to other things. There is some potential for utilizing partial pressure of venous oxygen (PvO_2) in the transport setting. PvO_2 samples are normally mixed-venous samples from a central line, but we could measure the partial pressure of oxygen from any blood source, to include venous blood from a peripheral venipuncture. Now blood on the venous side has already passed the capillary beds and therefore is dependent on delivery, metabolic need at the tissues, and offloading, so we would need to keep those things in mind. And in fact, there have been investigations into how we could use a peripheral blood gas to direct treatment if we do keep these other components in mind.[149] It's not that common at this point, but neither are arterial sticks in transport; using a PvO_2 could be a bridge to fill that gap.

One last summary before moving on from oxygenation. Oxygenation is one of the three big things in mechanical ventilation. We measure it via pulse oximetry, which tells us how filled up with oxygen the hemoglobin molecules (attached to red blood cells) in the blood are as they move past wherever we have attached the pulse oximetry probe. To get a better number (or improve oxygenation) by moving things around on the vent interface, we have three options and we typically do them in this order: increase FiO_2, add PEEP, and lengthen inspiratory time. All that said, let's not forget the basics: position the patient appropriately, verify that perfusion is adequate, and make sure ventilation and comfort are addressed simultaneously (see next sections).[150]

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[146] This is an oft-cited example in the critical care transport space to explain the oxygen-hemoglobin dissociation curve in clinical practice, but we have been unable to find any data on an actual clinical effect

[147] Hasudungan, 2018 - To dig more into this idea of what can affect hemoglobin's affinity for oxygen, refer to this video (and it also applies to the following section)

[148] Farkas, 2016; Farkas & Thomas, 2023 - This written piece outlines a number of situations in which pulse oximetry might be preferred to the arterial blood gas; the podcast discusses much of the same; and then see our discussion of **Blood Gas Analysis** for more

[149] Chemtob & Moller-Sorenson, 2018 - These guys looked into this concept of utilizing a PvO_2 with a peripheral venous sample to direct and guide treatment

[150] And to link to these other ideas: **Fraction of Inspired Oxygen**, **Positive End-Expiratory Pressure**, **Inspiratory Time (and I:E Ratio)**; **Ventilation**, **Comfort**

Ventilation

The next very important, big thing is ventilation. Ventilation refers to the movement of air in and out of the system as we both deliver breaths and allow exhalation. As discussed before, this is vitally important for the movement of carbon dioxide. Too much carbon dioxide hanging out in the lungs with no escape is bad news, so we can't just focus on getting oxygen in. And while we typically think of carbon dioxide in the context of acid-base analysis, recognize that there are a great many reasons to keep it well-controlled.[151] There are two strategies to determine if we are moving enough air for a given patient and we will discuss them both in turn: calculated minute volume and end-tidal monitoring of carbon dioxide ($EtCO_2$).

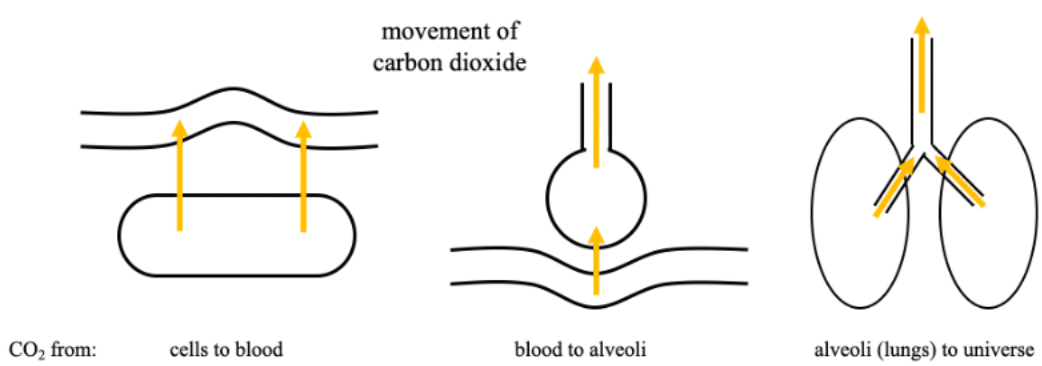
If we math it out, our minute volume goal for the typical adult patient should be:[152]

$$MV \approx 100ml/kg\ IBW/min$$

This number varies a bit for patients with an increased need (i.e., **Acidosis**), but it's a good place to start as written and is an appropriate minimum for most patients. Having a goal minute ventilation in mind and then assessing actual minute volume (typically measured by the vent and expressed as exhaled minute volume) is a great way to ensure that the patient's needs are met.

Concurrently, we also use $EtCO_2$ to monitor ventilation. When the body uses up oxygen at the tissue level, it kicks back carbon dioxide into the bloodstream. That carbon dioxide then makes its way up to the lungs where it passes into the alveoli and is then exhaled out. In theory, the value we see on our monitor for $EtCO_2$ is closely correlated with the arterial partial pressure of CO_2 ($PaCO_2$) and it is $PaCO_2$ that we are attempting to evaluate indirectly with $EtCO_2$. A few notes about all of this: $EtCO_2$ generally correlates with $PaCO_2$ fairly well, with $EtCO_2$ normally 2-5mmHg below $PaCO_2$. That normal difference is due to anatomic dead space and will increase with additional dead space (i.e., alveolar dead space).[153] That said, even with more dead space at play, $EtCO_2$ and $PaCO_2$ will move in stepwise fashion at the same rate.[154]

The whole process of CO_2 movement looks about opposite to our previous sketch showing how oxygen moves through the system:



The value we get on our quantitative $EtCO_2$ reading is a function of all of these factors. The standard approach to managing ventilation with $EtCO_2$ is to use a base range and adjust minute volume to get the value

[151] Yartsev, 2023g - See this page for a very thorough overview of how carbon dioxide can affect the body in different ways; also refer to the section on **Blood Gas Analysis** for more on that idea

[152] We discussed where this minute volume goal number comes from previously (in the section titled **Minute Volume**); and see section on **Ideal Body Weight** to review the basis of this idea, that lung size best correlates with height; and then, as noted before, see **More on Pediatric Ventilation**, for the kiddo-specific stuff

[153] Yartsev, 2025a - Provides an in-depth discussion of this and all the specific things that can result in alveolar dead space and a subsequent gap between $EtCO_2$ and $PaCO_2$.

[154] Siobal, 2016 - And look here for more information on carbon dioxide monitoring in general; and see **Blood Gas Analysis** for an in-depth discussion on this gap between $EtCO_2$ and $PaCO_2$

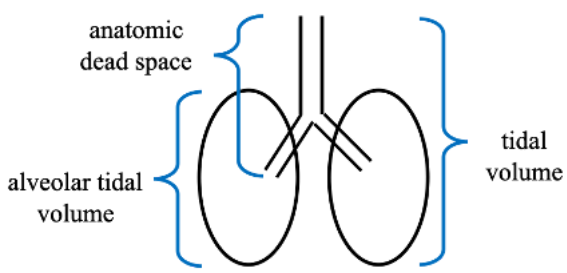
within that acceptable range. Normal range for $EtCO_2$ is 35-45mmHg; values above range require an increase in minute volume to blow off more carbon dioxide, values below that require us to read the next paragraph carefully.

A low $EtCO_2$ can be caused by a few different things, one of which is hyperventilation. This can be detrimental to a patient, as an alkalotic state (due to too much minute volume and a low $EtCO_2$) can throw off the patient's homeostasis.[155] In this case, it'd make sense to decrease minute volume (by lowering either rate or tidal volume) to get the $EtCO_2$ (and therefore ventilation) back to normal. All that said, a low $EtCO_2$ could also be due to a breakdown somewhere else in the system (i.e., at any of those yellow lines in the previous drawing). For example, if perfusion is no good (i.e., shock) we may see a low $EtCO_2$ even though the issue is not necessarily a ventilation problem. In this case we could kill the patient by chasing their $EtCO_2$ or dropping minute volume to an unsustainable level.

We can navigate this whole situation by managing ventilation by looking at both minute volume and $EtCO_2$ instead of just $EtCO_2$. There are times when we will be a bit off with minute volume and others when our goal range for $EtCO_2$ varies, but this system of dual parameters to evaluate ventilation is a safety check to remind us of all the factors involved. To summarize: we measure ventilation using both a calculated minute volume goal and $EtCO_2$. This minute volume goal, which is considered a minimum in most cases, is around 100ml/kg IBW/min. Normal $EtCO_2$ is 35-45mmHg, but baseline for an acutely ill patient may be at the low end of that range or lower.

And one final point before we move on: when faced with the choice as to whether we should manipulate **Respiratory Rate** or **Tidal Volume** to effect a change in **Minute Volume**, here's what we recommend: to increase minute volume, utilize tidal volume first; to decrease minute volume utilize rate first. Now we are going to outline an argument as to why that is and then follow up with some thoughts on why that rationale isn't quite as good as it is often presented to be. So hold with us through this explanation and then we'll discuss it afterwards. Know that the basis of the idea still holds true, it's mainly a matter of to what degree this difference actually exists in practice.

Let's say we have a patient breathing at a rate of 15 and a tidal volume of 450ml:



tidal volume 450ml
- anatomic dead space 150ml
---
alveolar tidal volume 300ml

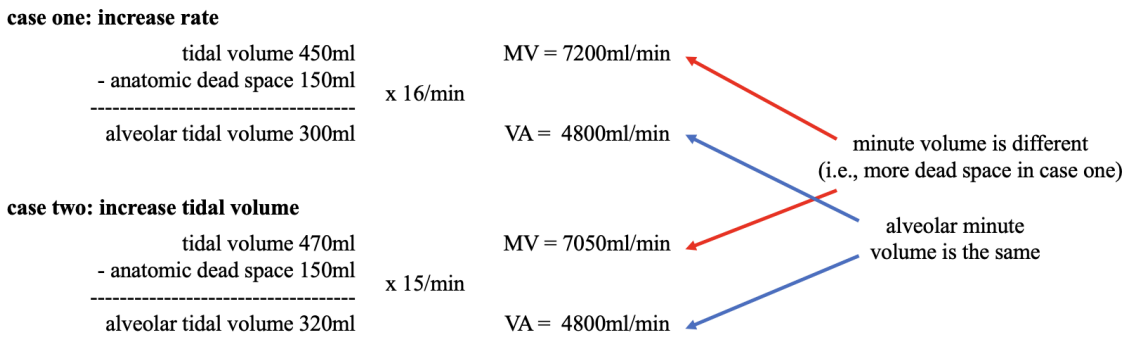
x 15/min

minute volume = 6750ml/min

alveolar minute volume = 4500ml/min

[155] That said, this is generally AOK unless taken to an extreme level (i.e., a low $EtCO_2$ due to excessive ventilation; a low $EtCO_2$ due to poor perfusion, on the other hand, is a bonafide problem)

Now assume we want to increase alveolar minute volume by an arbitrary value of 300ml. We could do this by either of two ways: increasing rate to 16 or increasing the volume of each breath by 20ml. While either method is just fine mathematically, adding in an extra breath is a bit less efficient and puts more stress into the system. That stress comes in a few different forms, all discussed elsewhere.[156] And here's how the math would look in either case:



Now on the opposite end of things, if $EtCO_2$ is low (which indicates too much minute volume), then we back off on respiratory rate first. That gives us the same differences, but in the reverse: less alveolar minute volume (which is what we want) accompanied by less of the negative consequences. In reality, either strategy (titrating rate or tidal volume) is appropriate, it's just an extra thing to keep in mind if we want to be more resourceful in our vent changes.

While we've shown just now that it's more efficient to use tidal volume to increase ventilation and rate to decrease ventilation, know that both anatomic and alveolar dead space can sometimes increase with more tidal volume.[157] This means that the difference in both dead space and alveolar minute volume in the above calculations may not be as marked as we've demonstrated. The tough part about this is that we can't rightfully predict to what degree dead space will track with an increase in tidal volume, as there isn't a direct relationship between the two and it depends on a few other things: how the current tidal volume compares to overall lung capacity, how the breaths are being delivered, other vent settings (especially PEEP), concurrent disease processes, etc. So while we still recommend to consider the difference and use some discretion when making changes, know that this argument doesn't give the whole picture and that we can utilize rate or tidal volume to change minute volume in either direction.

And to summarize: ventilation is another one of the big things to address in mechanical ventilation. We start by using a weight-based goal for minute volume (by way of an age-based respiratory rate and weight-based tidal volume) and then titrate it as we go to an $EtCO_2$ goal. This minute volume goal is 100ml/kg IBW/min and a normal $EtCO_2$ is 35-45mmHg. More minute volume will drive $EtCO_2$ down, less minute volume will lead to an increase in $EtCO_2$. To increase minute volume, we go up on tidal volume and then rate; to decrease minute volume, we go down on rate and then tidal volume.

[156] More breaths means more of an impact on hemodynamic function (discussed in both **How is Positive-Pressure Ventilation Different?** and **Hypotension**) and an extra inflation/ deflation cycle which can put stress on the alveoli (discussed already in **Positive End-Expiratory Pressure** and again later on in **Driving Pressure**)

[157] Yartsev, 2023a - This page discusses the variation in dead space with changes in tidal volume; also refer back to the section on **Dead Space** if need be

Comfort

The third very important parameter to consider with vent management is patient comfort. A comfortable patient who is breathing in coordination with the ventilator receives the full therapeutic benefit of the intervention. A disruption in this coordination (i.e., discomfort) means that the desired effect is attenuated or diminished to some degree. Discomfort also presents a risk of injury; this could be due to a **Minute Volume** that strays too high or too low, airway pressures that get too high, and/ or psychological distress from the experience of it all. In this section we'll talk about general concepts related to assessing and providing patient comfort; we'll defer a more detailed discussion of **Asynchrony & Dyssynchrony** for when we get to **Vent Graphics**.

As for a bit of background, discomfort in mechanical ventilation involves a number of distinct, but interrelated concepts. First, and likely most familiar, is pain - this can be related to the process of intubation and mechanical ventilation itself or the underlying injury/ illness for which the thing was done in the first place. Next is dyspnea or a subjective, distressing sensation felt by the patient related to how he or she is being ventilated, often exacerbated by being unable to communicate and/ or control breathing independently.[158] And then lastly is ventilator dyssynchrony which is when the vent itself is competing with or interfering with the patient's effort to breathe. Managing all of these things concurrently can be complicated, but it is not impossible. We'll try to lay out a framework for working through it all systematically.

To start with, it is important to have a strategy for quantifying or measuring comfort so that we can gauge the efficacy of our interventions. Many agencies recommend scales or tools to use and here are some examples:

TABLE 3. RICHMOND AGITATION–SEDATION SCALE

Score	Term	Description
+4	Combative	Overtly combative or violent; immediate danger to staff
+3	Very agitated	Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff
+2	Agitated	Frequent nonpurposeful movement or patient–ventilator dyssynchrony
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	
−1	Drowsy	Not fully alert, but has sustained (more than 10 s) awakening, with eye contact in response to voice
−2	Light sedation	Briefly (less than 10 s) awakens with eye contact in response to voice
−3	Moderate sedation	Any movement (but no eye contact) in response to voice
−4	Deep sedation	No response to voice, but any movement in response to physical stimulation
−5	Unarousable	No response to voice or physical stimulation

Procedure

1.	Observe patient. Is patient alert and calm (score 0)?
	Does patient have behavior that is consistent with restlessness or agitation (score, +1 to +4 using the criteria listed under Description)?
2.	If patient is not alert, in a loud speaking voice state patient's name and direct patient to open eyes and look at speaker.
	Repeat once if necessary. Can prompt patient to continue looking at speaker.
	Patient has eye opening and eye contact, which is sustained for more than 10 s (score, −1)
	Patient has eye opening and eye contact, but this is not sustained for 10 s (score, −2)
	Patient has any movement in response to voice, excluding eye contact (score, −3)
3.	If patient does not respond to voice, physically stimulate patient by shaking shoulder and then rubbing sternum if there is no response to shaking shoulder.
	Patient has any movement to physical stimulation (score, −4)
	Patient has no response to voice or physical stimulation (score, −5)

Reprinted by permission from Reference 105.

[158] Kemoun & friends, 2025 - Solid overview of dyspnea in mechanical ventilation

TABLE 1. NONVERBAL PAIN SCALE

	Category		
	0	1	2
Face	No particular expression or smile	Occasional grimace, tearing, frowning, wrinkled forehead	Frequent grimace, tearing, frowning, wrinkled forehead
Activity (movement)	Lying quietly, normal position	Seeking attention through movement or slow, cautious movement	Restless, excessive activity and/or withdrawal reflexes
Guarding	Lying quietly, no positioning of hands over areas of body	Splinting areas of the body, tense	Rigid, stiff
Physiological I (vital signs)	Stable vital signs (no change in past 4 h)	Change over past 4 h in any of the following: SBP > 20 mm Hg, HR > 20 beats/min, RR > 10 breaths/min	Change over the past 4 h in any of the following: SBP > 30 mm Hg, HR > 25 beats/min, RR > 20 breaths/min
Physiological II	Warm, dry skin	Dilated pupils, perspiring, flushing	Diaphoretic, pallor

Definition of abbreviations: HR = heart rate; RR = respiratory rate; SBP = systolic blood pressure.

Reprinted by permission from Reference 15.

Note that there are separate scales for pain and sedation. Pain scales are, obviously, for pain; sedation scales sort of assess comfort in a more general sense. It is often the case that these concepts blur together in mechanical ventilation, especially since pain, dyspnea and dyssynchrony can all present similarly. Also many of the **Agents for Comfort and/ or Synchrony** have both analgesic and sedative effects. That said, they are distinct concepts and it helps to both assess/ evaluate and treat them independently. There are also scales to measure dyspnea, but they overlap quite a bit with the pain and sedation scales (which are more common in transport), so it may not make sense to utilize all three.[159]

Goals for pain are comparable to those for non-ventilated patients, basically whatever the patient tolerates. The difference, obviously, is that we often cannot get an answer from the patient about how they feel subjectively. Utilizing a minimum acceptable score *and* ruling out dyspnea and/ or dyssynchrony is a reasonable starting point. Goals for sedation of mechanically ventilated patients in transport have traditionally been towards the "heavy sedation" or "zero movement" end of the spectrum, but this has been changing over the years. On the hospital side, there has been a significant shift towards light sedation, but that has been slow to cross over into transport due to concerns cited about safety and risk of agitation while in motion. That said, so long as there is no evidence of dyspnea and/ or dyssynchrony, it is generally safe to assume that sedation is adequate.

Carrying on with this idea, a completely sedated patient making no effort to breathe on his or her own suffers the negative consequences of positive-pressure ventilation.[160] Maintaining patient effort and supporting it appropriately with the machine decreases the degree of those negative things we previously discussed and helps improve both **Ventilation** and **Oxygenation**.[161] Furthermore, having access to subjective feedback from the patient (effort to breathe, movement, response to stimuli, etc.) allows us to better monitor whatever else is going on. Because of this, sedation to the point of no spontaneous effort (or even paralysis, for that matter) should be a last resort for nearly all ventilated patients; instead, we should attempt to maintain comfort to a controlled level using both analgesic and sedative medications (which, just to clarify, are two distinct things).[162]

To be clear, there are some patients who will need to be completely sedated (i.e., "deep sedation" or "unarousable") and/ or paralyzed. In the transport setting, this often comes up in the context of safety concerns. A patient who has previously been difficult to restrain or who has already self-extubated once before may need

[159] One such scale is the Respiratory Distress Observation Scale (RDOS)

[160] We discussed these consequences of mechanical ventilation in **How is Positive-Pressure Ventilation Different?**

[161] Mauri & friends, 2017; Macintyre, 2014 - The first discusses how to navigate the benefits of spontaneous breathing in the ventilated patient with potential consequences; the second outlines how comfort can decrease oxygen consumption

[162] Patel & Kress, 2011 - This article outlines some thoughts on both analgesia and sedation in mechanical ventilation, we discuss these things further in **Medications in Mechanical Ventilation**; we've also taken the graphics for the NVPS and RASS scores from this article

to be paralyzed to ensure safe transport. There may also be the argument that breathing spontaneously or making the effort to do so increases oxygen consumption and could exacerbate certain conditions. So while we generally prefer not to paralyze anyone, it may very well be in his or her best interest for us to do so. Just remember that we will still need to consider discomfort (and pain in particular) and address it appropriately. In those cases, we can make a subjective impression about comfort by looking at physiologic changes as outlined on the bottom two lines of the NVPS score:

TABLE 1. NONVERBAL PAIN SCALE

	Category		
	0	1	2
Physiological I (vital signs)	Stable vital signs (no change in past 4 h)	Change over past 4 h in any of the following: SBP > 20 mm Hg, HR > 20 beats/min, RR > 10 breaths/min	Change over the past 4 h in any of the following: SBP > 30 mm Hg, HR > 25 beats/min, RR > 20 breaths/min
Physiological II	Warm, dry skin	Dilated pupils, perspiring, flushing	Diaphoretic, pallor

Definition of abbreviations: HR = heart rate; RR = respiratory rate; SBP = systolic blood pressure.

Now let's move on to differentiate between the ideas of synchrony and compliance. Synchrony is when the ventilator's efforts are in line with the patient's respiratory effort. Dyssynchrony, therefore, would be when the patient wants a breath in a given instant, but the machine decides to give a breath some other time.[163] Compliance, on the other hand, is used to describe how well the patient follows the lead of the ventilator.[164] A paralyzed patient is for sure compliant, but that doesn't mean it should be the goal we aim for. Rather we should strive for synchrony and let the patient take the lead on things, adjusting settings along the way to match mechanical support to patient cues.

The strategy to address comfort for the ventilated patient is to treat the extreme end of discomfort using drugs (both analgesics and sedatives, discussed more in **Medications in Mechanical Ventilation**) and then do what we can to optimize synchrony on the vent itself once the patient is comfortable enough to respond to more fine-tuned settings. To begin this fine-tuning, we first need to make sure that patient effort to breathe is supported by the machine. We'll talk about **Triggers** later, but the general idea is that we don't want to ignore patient effort and we also don't want to send breaths down the circuit accidentally. We may have to trial different thresholds and types of triggers until we find what best suits the patient and that's completely OK.

Another thing we can do is adjust **Inspiratory Time (and I:E Ratio)**. Occasionally a minor adjustment here can make a patient feel more comfortable. One thing to keep in mind is that a normal I:E ratio is 1:2 and that a larger ratio closer to 1:1 is common at exercise or exertion.[165] While the link from exercise to acute illness may not hold up in all cases, this helps explain how the lower ratios (1:3 and beyond) we commonly end up with on the vent (due to how settings get auto-calculated) may predispose our patient to discomfort. For example, if we start with an I:E ratio of 1:2 and then titrate rate down to accommodate alow $EtCO_2$ and don't simultaneously increase inspiratory time, we will have a smaller I:E ratio (again, 1:3 and beyond) that can be uncomfortable for our patient.

Switching mode or control may also help with discomfort. We mentioned this already, but different **Types of Breaths** are delivered differently in each mode or method of control, and sometimes one may work better for the patient. Discomfort may be a valid reason to switch from volume control to pressure control (or vice versa) or to move from **Synchronized Intermittent Mandatory Ventilation** to **Assist Control** (again, or

[163] The term "asynchrony" is also used quite a bit in these sorts of discussion, we'll stick with dyssynchrony here and explain this a bit more in **Asynchrony & Dyssynchrony**

[164] And this use of the term compliance to describe a patient following the ventilator's lead is different than the idea of lung compliance which we discuss later in **Compliance & Resistance**

[165] Johnston, 2017 - This page looks at this concept of I:E ratio with exertion in terms of a fraction of inspiratory time over expiratory time; while it is geared to pulmonary function tests and exercise physiology, it is still applicable to our discussion

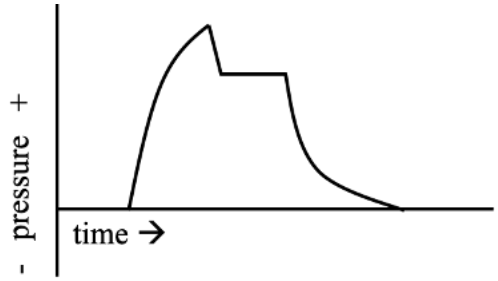
vice versa; although going from AC to SIMV is much, much less common).[166] And specifically for SIMV, if we have a patient triggering breaths we can go up on pressure support and see how (s)he responds. We mentioned already that it is the custom to have pressure support breaths be smaller than mandatory or assisted ones, but that doesn't have to be the rule.

Now let's summarize our approach to patient comfort, the third of the three big things to be addressed for all ventilated patients. Our goal in addressing comfort is to maximize synchrony and this includes both matching ventilator effort with patient need and maintaining the patient's spontaneous effort. To help gauge this all, a scoring system should be utilized. Extremes of discomfort get treated pharmacologically with both analgesics and sedatives. After that, however, we can fine-tune ventilator settings to further maximize efficacy. Specific strategies include adjusting triggers, increasing inspiratory time to yield to a higher I:E ratio, trialing a different mode or method of control, and using higher values of pressure support if in SIMV mode.

[166] And refer back to **Modes of Ventilation** and **Control of Ventilation** to brush up on these ideas

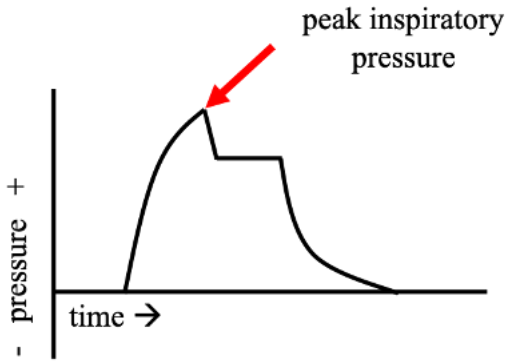
Vent Parameters Round Two

This next section discusses a few more vent parameters that we measure after the initial setup or taking over of a ventilated patient. They are considered separately from the values previously discussed because they are dependent on other things. We don't dial them into the machine, but rather we measure them to assess how things are coming along with the values we are able to control. To help clarify these ideas, let's refer back to an image we previously discussed. It shows pressure we put into the system over time as a breath is delivered in volume control ventilation:



We used this graphic before to demonstrate a couple of things, but it is now worth mentioning that this waveform and the two subsequent concepts (peak inspiratory pressure and plateau pressure) apply to volume control ventilation. Let's first get things clarified for volume control ventilation, and then we'll talk about how these ideas carry over into pressure control ventilation.

Peak Inspiratory Pressure[167]



Peak inspiratory pressure (PIP) is the highest point on this waveform. It represents the maximum pressure as we deliver a breath into the system. It is also known as peak pressure (Ppeak). Peak inspiratory pressure is a function of both how we deliver a breath via the machine and how easily that breath can get from the machine down to the lungs. A normal peak inspiratory pressure is less than 35cmH_2O. An isolated peak inspiratory pressure that is too high generally won't cause damage to the patient, rather it likely indicates something gone wrong in the system. This is particularly relevant when we have a normal peak pressure that then becomes elevated. In these cases it is important to seek out the cause and fix the underlying issue.

On the machine end, peak inspiratory pressure is the result of **Flow**, which (recall from our previous discussion) essentially describes how fast we push air to achieve a breath. We sometimes can't manipulate flow directly on transport ventilators, so to decrease peak inspiratory pressure by pushing buttons on the machine we have to make things happen in a roundabout way. Which isn't ideal and the truth of it all is that most of the

[167] Nickson, 2019a - Short article that provides another good review of both peak inspiratory pressure (this section) and plateau pressure (next section); cited normal values are supported by his content

peak inspiratory pressure issues we face are due to pathophysiology or equipment issues, so let's just skip right on ahead to how we can decrease peak inspiratory pressure via other mechanisms outside of the vent itself.[168]

Causes of an elevated peak inspiratory pressure include things like secretions in the endotracheal tube, bronchospasm, patient discomfort, mainstem intubation, pneumothorax, pulmonary edema, etc. Any time we see a high peak inspiratory pressure we ought to try and identify a cause.[169] Once that cause is identified, we can then decide whether or not an action is needed. For example, a high peak pressure due to secretions should get suction and a high peak pressure due to a pneumothorax may lead to decompression (i.e., if tensioning); on the other hand, a high peak pressure due to a small endotracheal tube may be acceptable. The peak inspiratory pressure in this last case represents an impediment to airflow due to the tube and not the patient's anatomy, so we may decide to leave it alone (especially if there is good reason for that small endotracheal tube, such as airway swelling).

Another consideration here is patient comfort and the idea of laminar flow. Without getting too far into this, recognize that air can move freely and efficiently through a uniform pipe or tube, but with movement or disruption to that tube, airflow will be more chaotic and result in higher pressures. Keeping our patient comfortable and in sync with the vent leads to more uniform (i.e., efficient) air movement and lower peak pressure. Takeaway here: make sure our ventilated patient is comfortable. And if we notice an increase in peak inspiratory pressure, **Comfort** ought to be one of the things to consider.[170]

To measure peak inspiratory pressure we simply need to look at the vent display. Most machines will either give us the value or show a barometer that fluctuates with each breath. Peak inspiratory pressure is always the highest value that comes up during a breath. Another way to keep an eye on peak pressure is by setting an alarm so that the machine yells at us when a certain pressure is reached. That said, there is one critical thing to know about this: yes it will tell us that the pressure has gotten too high, but it will likely (depending on model) also cycle off the breath it is giving in response to that high-pressure alarm. This can potentially kill our patient and we will get into that a bit more later on.[171]

So in summary, peak inspiratory pressure represents the maximum pressure as a breath is delivered by the machine. A normal value is less than 35cmH_2O and we measure it by looking at the feedback on the vent interface. Potential causes include too much air, too much flow, small or kinked endotracheal tube, patient discomfort, secretions, pneumothorax, mainstem tube placement, and bronchospasm. Troubleshooting these sorts of problems will be reviewed again in **Keeping Things Going**.

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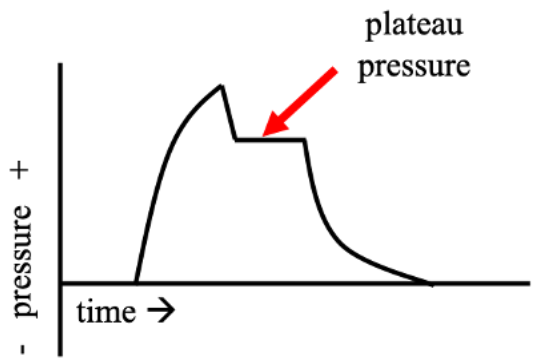
[168] But for the curious folks out there: in volume control flow is determined from tidal volume and inspiratory time; in pressure control (and with pressure support breaths) it is a function of pressure and rise time

[169] And one part of how to identify the cause of a high peak inspiratory is by assessing **Plateau Pressure** (next section); we have this all drawn out in a flowchart later, but first we need discuss all the terms and concepts first (see **Watching Pressures**)

[170] And then a bit more specifically on the **Comfort** comment - consider **Asynchrony & Dyssynchrony**, as discussed in **Vent Graphics**

[171] This discussion of how breaths can get terminated early with the triggering of a high-pressure alarm is in the section on **Alarms**

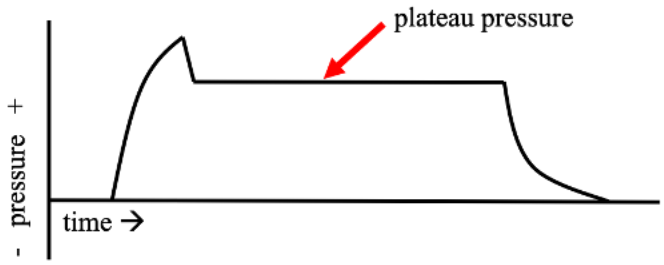
Plateau Pressure



Plateau pressure (Pplat) is the pressure in the system once the lungs are filled with air and before the breath cycles off. It represents the average pressure at the alveoli as they are at maximum inflation during inhalation. A normal plateau pressure is less than 30cmH_2O. Values higher than that can lead to direct damage to the alveoli, which can subsequently cause issues with pulmonary respiration.[172] There is no minimum or lower limit for plateau pressure, but recognize that lungs that aren't being filled all the way (i.e., a low plateau pressure) may not be maximizing the surface area of alveoli and therefore **Oxygenation** may not be at its best.[173]

The primary cause of a high plateau pressure at the start of ventilation is too much **Tidal Volume**. That said, it can also be present or develop over time due to patient discomfort, mainstem migration of the endotracheal tube, atelectasis, and pulmonary edema. If we get a high plateau pressure, consider these other causes (and address them appropriately) before dialing down tidal volume, as we don't want to give up ventilation unnecessarily.[174] We do, however, want to avoid a sustained high plateau pressure over many breaths, as that can lead to damage to the alveoli.

Some modern machines will simply display the plateau pressure on the vent monitoring screen with no extra legwork involved, but on older or more simple machines, a maneuver called an inspiratory pause is required. Basically we deliver a breath and then instead of letting the breath cycle off to exhalation, we simply hold that inspiration at a constant pressure and see what it is. It would look something like this:



We perform the inspiratory hold maneuver (in whatever way is appropriate for our particular machine) and the plateau pressure either pops up on the screen for us or we have enough time to read the value from the barometer. There isn't a universally accepted frequency for measuring this (or any of the other pressures discussed in this section), but it seems to make sense that we just add them on to our reassessment of vital signs (so every 10-30 minutes, depending on the transport program and patient acuity). While that may be overkill, it's better to measure too much than to miss things due to not checking often enough. At a minimum, plateau pressure should be measured after any increase in tidal volume to make sure that we don't cause alveolar damage (and this includes after first putting the patient on the vent).

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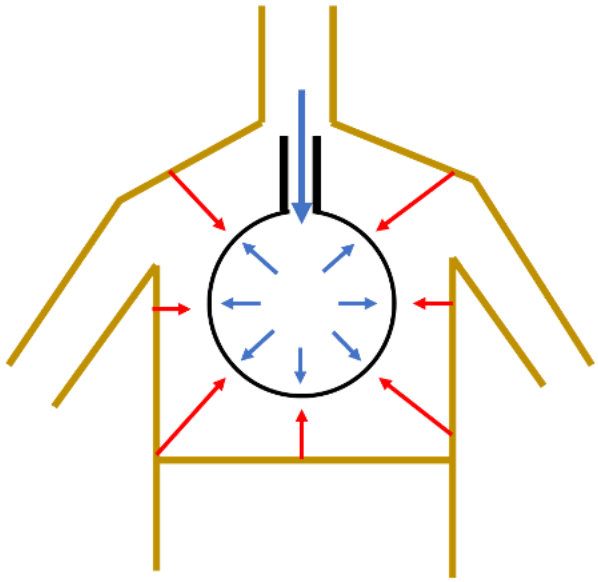
[172] We defined this concept of pulmonary respiration back in **Terms to Describe Breathing**
[173] In **Titrating Up on Tidal Volume?** we will review thoughts on how to address a low plateau pressure
[174] We revisit this idea of managing abnormal airway pressures in an algorithmic fashion in the section called **Watching Pressures**

Another thing about plateau pressure is that the value we get is an average of alveolar pressures across the lung - some regions will experience higher pressures and others will experience lower pressures. The lung is not uniform throughout, but we can't measure alveolar pressures in specific lung regions or see to what degree this value would vary across the different parts. The safe limit of less than 30cmH_2O is a good guideline by which to limit our vent settings, but recognize that this doesn't mean that a pressure higher than that to one alveolus or a region of the lung will always cause harm. Likewise, a plateau pressure below 30cmH_2O is not a guarantee that damage will not be caused.

One more subtlety here is that an elevated plateau pressure doesn't always reflect stress on the alveoli, there may be something external to the alveoli that prevents them from opening:



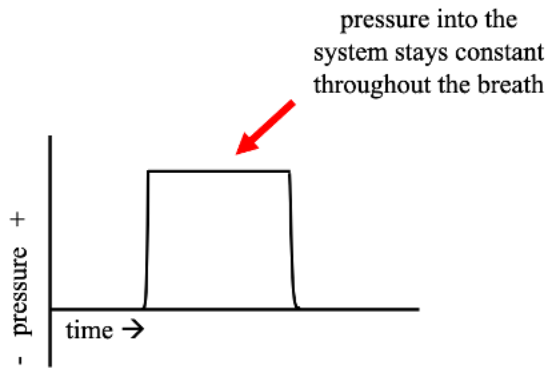
In these cases the elevated plateau pressure is not due to distention at the alveoli, rather it is due to something else. Examples would be a tension pneumothorax, burn to the chest wall, or even physical compression as with an entrapped patient. So while we generalize plateau pressure as a reflection of alveolar pressure, know that this isn't always the case.

In summary, plateau pressure is typically the pressure seen by the alveoli when we deliver a breath in volume control ventilation. A normal value is less than 30cmH_2O and we measure it by performing an inspiratory hold maneuver. While there is no bottom limit to plateau pressure, it is important to recognize that we want to fill the lung and alveoli up with each breath delivered, so be wary of a super low plateau pressure and consider inadequate tidal volume (and subsequently minute volume). High plateau pressure can be caused by too much tidal volume, pneumothorax, restriction to chest wall expansion, mainstem intubation, and a few other things that we'll spell out later on.[175]

[175] This will be in **Watching Pressures** and in **Acute Deterioration**

Airway Pressures in Pressure Control?

Up to this point we've discussed **Peak Inspiratory Pressure** and **Plateau Pressure** in the context of **Volume Control** ventilation, but things are a bit different in **Pressure Control**. Let's start with what a pressure control breath looks like mapped out as pressure over time:[176]



First thing to mention here is that the top of that waveform (i.e., the flat part) represents the value we have set for pressure control and that we often refer to that value as peak inspiratory pressure. For example, we could either say that the patient is in pressure control with a pressure control of X or that the patient is in pressure control with a peak pressure of X. Either way is just fine. The only caveat here is to consider if that pressure control value is additive to or inclusive of PEEP (as we discussed in **Types of Breaths**). Peak inspiratory pressure is always an absolute value that represents a pressure measured against the atmosphere.

Since the machine in pressure control limits pressure into the system, the actual highest pressure will only be above that flat line at the top of the square waveform (marked by the red arrow in the graphic) if something causes a disturbance in what the machine is doing, such as a hiccup, patient movement, speedbump, etc. The machine won't intentionally put more pressure than what we have set, but a peak pressure higher than the set pressure control can occur. So while we may still set a high-pressure alarm and monitor peak inspiratory pressure in pressure control ventilation, our concern is more for being aware of disturbances to the system rather than being aware of changes to airflow, as was the case in volume control ventilation.[177]

Next thing: the goal is that the average alveolar pressure eventually does equal that pressure represented by the top of the square waveform (towards the end of expiration), therefore we assume it to be true that pressure control equals plateau pressure.[178] And because of this assumption that mostly holds true, it's OK that some machines don't let us do inspiratory holds in pressure control ventilation, as the data gleaned from the test just wouldn't provide any additional information. Also because the primary reason we want the plateau pressure (in volume control) is to rule out high alveolar pressures (to ensure the wellbeing of the alveoli); in pressure control if plateau pressure doesn't match pressure control it's because true plateau pressure is less than the pressure control (which is a bummer, but not a safety concern for the alveoli).

Now the way it works is that it takes time for the alveolar pressure to rise up to match the pressure going into the system. Even though we start with a high pressure at the machine end of the system, it may take some time for that pressure to equalize down to the alveoli. If our **Inspiratory Time** isn't long enough to allow that to happen, the alveolar pressure may not ever get up to the level we have set for pressure control. We work around that in volume control by performing an inspiratory hold and waiting for as long as we need to in order to see that pressure even out. We don't always do that in pressure control because, as we said just a moment ago, the **Plateau Pressure** won't be above our pressure control value and it isn't so much of a concern.

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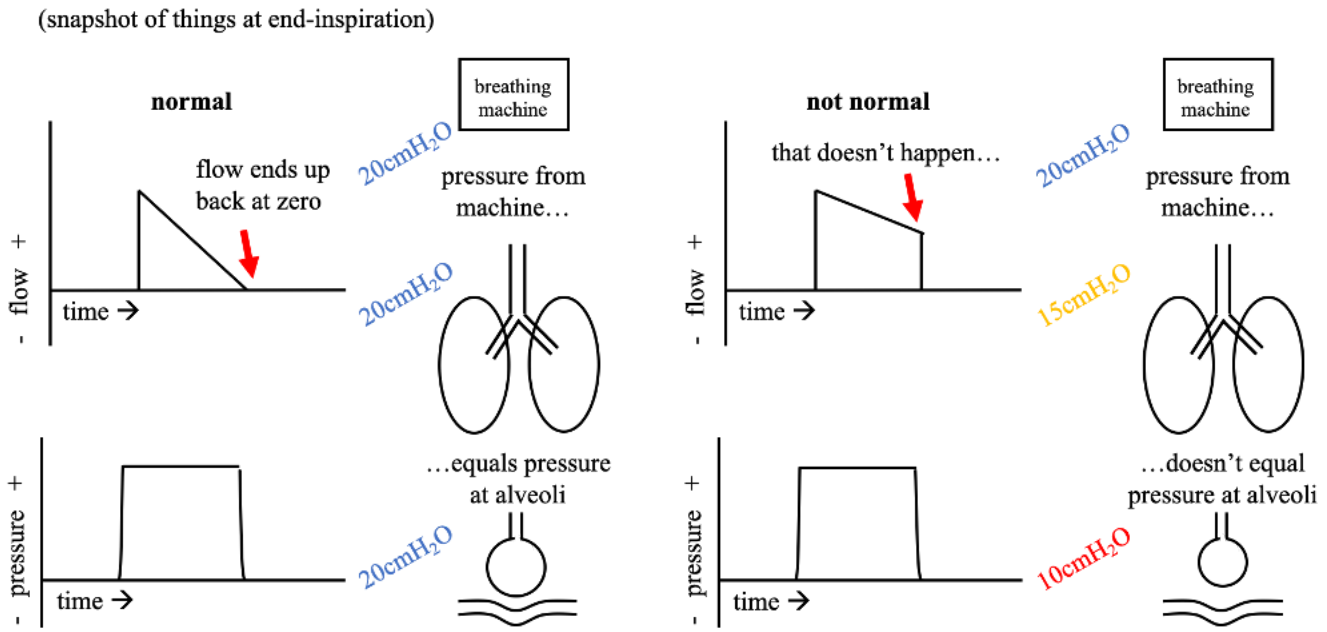
[176] To review these types of waveforms and how this pressure control waveform differs from a volume control one, refer back to **Types of Breaths**

[177] In pressure control ventilation, we become aware of those obstruction issues by monitoring exhaled tidal volume (and maybe flow, if available on our particular machine); also see **Alarms** for a more detailed discussion of this piece

[178] Hess, 2014 - Another way to say this is that if flow gets to zero during the inspiratory phase, then peak inspiratory pressure equals plateau pressure

But if we wanted to know a little more about what's going on in the alveoli and we can't do an inspiratory hold on our machine in pressure control, we can get a partial picture of things by looking at the flow waveform.[179] Pressure control breaths start with a higher flow that then drops down towards zero throughout the breath. While it may be hard to see with quantitative values on our machine (unless we can view waveforms), if flow doesn't get down to zero before the breath cycles off, then we can consider that the pressure in the alveoli may not have made it up to the level we put in on the front end:



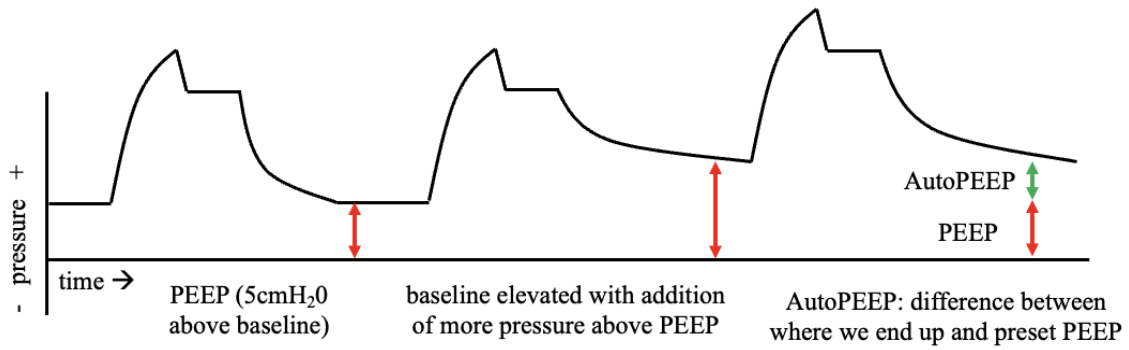
All that said, this isn't a great method unless we have waveforms to look at. And even then it's a binary thing. It says whether or not alveolar pressure got up to the value of pressure control, but it doesn't tell us what the alveolar pressure actually was. There are other ways to measure or approximate plateau pressure, although they are unlikely to be available to us in the transport setting.[180]

So what utility is there in knowing plateau pressure in pressure control anyways? We said already that the usefulness of this information in volume control is to prevent damage to the alveoli, but that isn't as much of an issue in pressure control. Potential uses of knowing a plateau pressure in pressure control would be making sure our inspiratory time is appropriate (i.e., that the inspiratory time is long enough to allow pressure going in to match pressure at the alveoli) and calculating things like compliance and driving pressure (both discussed later).[181] These are all cool things to work with, but it takes both time and effort and, therefore, may not be the best use of one's cognitive capacity when managing a sick patient in the transport setting. We will discuss this stuff, but know that plateau pressure is primarily a tool for ensuring alveolar safety in volume control ventilation.

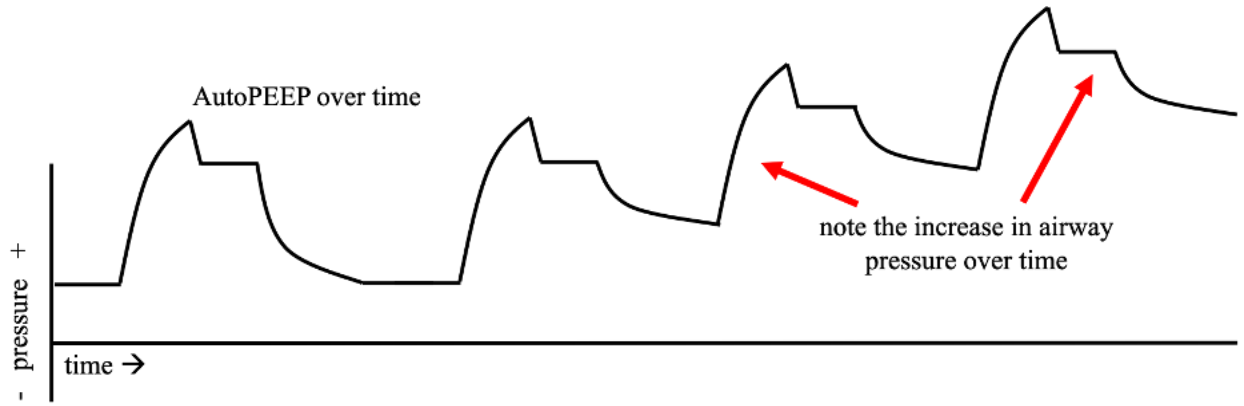
[179] See **Vent Graphics** for more on this
[180] Mojoli & friends, 2015 - This short paper assesses the efficacy of these alternative methods of measuring plateau pressure
[181] In the sections **Compliance & Resistance** and **Driving Pressure**

AutoPEEP

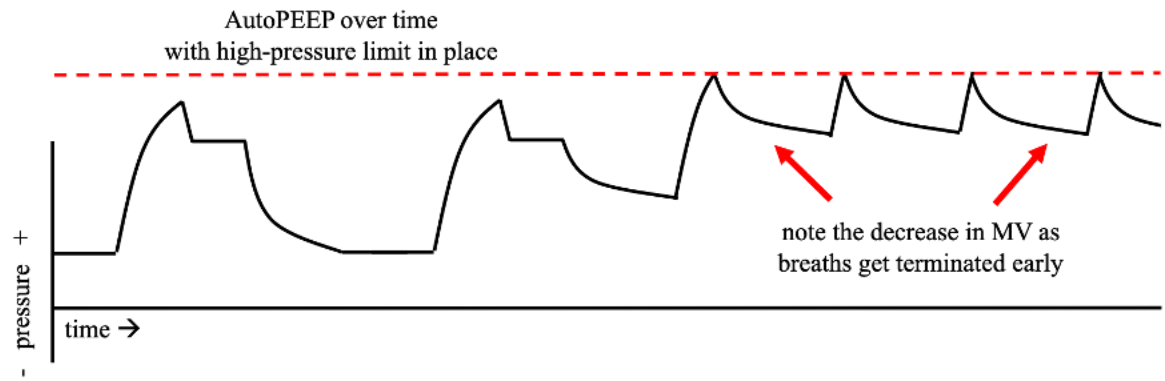
AutoPEEP is the idea of **Positive End-Expiratory Pressure** being added into the system inadvertently. It is also known as intrinsic PEEP. We said before that atmospheric pressure is assumed to be 0cmH_2O as the starting point for vent discussions and that PEEP is the addition of pressure on top of that (i.e., "adding 5cm of PEEP" to reset that baseline to 5cmH_2O). With that in mind, AutoPEEP is when that baseline starts to creep up from whatever we have set as PEEP to higher values because the patient isn't able to exhale all the way back to baseline before the next breath comes around. This idea is commonly referred to as breath stacking and can be represented like this:



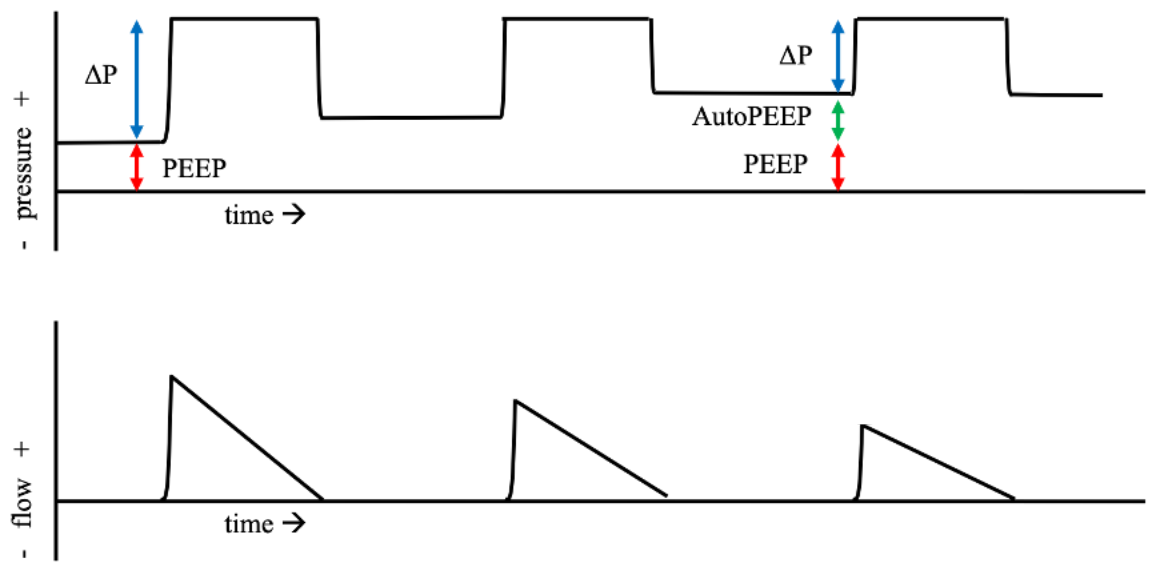
Normal AutoPEEP is zero, which means there shouldn't be any AutoPEEP in the system at all. That said, a few cmH_2O worth of AutoPEEP may be OK before we take action. Presence of AutoPEEP in **Volume Control** can lead to an increase in other airway pressures, most importantly of which is **Plateau Pressure**:



Another thing to realize is that if we have a normally-set high-pressure limit in place, then **Minute Volume** will suffer as breaths get terminated early:[182]



AutoPEEP in **Pressure Control** will also result in decreased minute volume (due to less exhaled tidal volume per breath), but by a slightly different mechanism. Breaths don't get cut short as they do in volume control, rather the flow to get to that set pressure is less. And since volume delivered is the product of flow and time, we get less volume:[183]



tidal volume = area under the flow time waveform
less ΔP = less flow required = less tidal volume

To measure AutoPEEP or to check its presence, it is sometimes necessary to perform another maneuver called an expiratory hold.[184] Doing an expiratory hold allows us to accurately see what the pressure actually is when we expect the breath to have returned to baseline. Normally the machine will calculate an AutoPEEP for us by subtracting PEEP from whatever pressure it measures during the hold.

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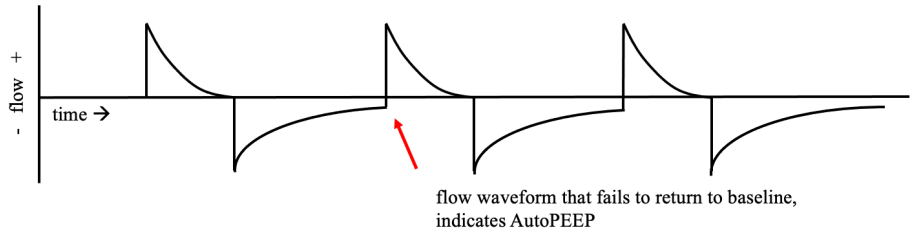
[182] We talk about this high-pressure alarm and the fact that it can compromise minute volume later on in **Alarms**

[183] We used the symbol ΔP to represent the difference between pressure control and (Auto)PEEP, this is also the notation for a concept known as **Driving Pressure** which we will get to in just a bit

[184] There are other ways to check for AutoPEEP, but they aren't typically available in transport unless we have access to waveforms

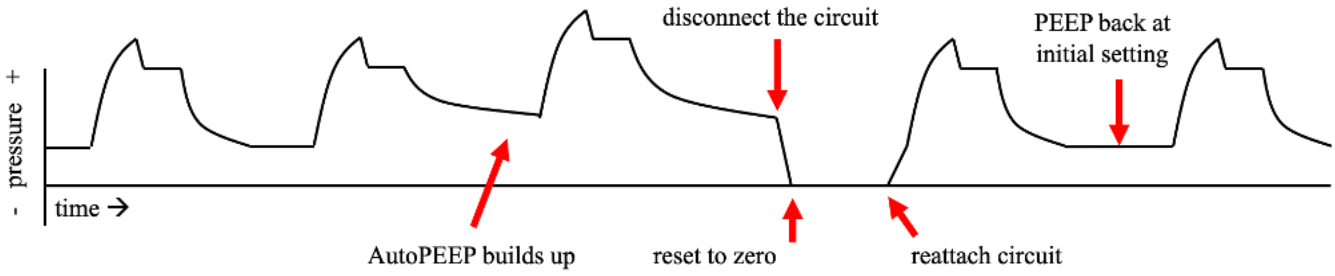
If our ventilator has the ability to show **Vent Graphics**, we can also look at the flow waveform or the flow-volume loop to check for the presence of AutoPEEP:[185]



And then some vents calculate AutoPEEP automatically and in real time, so there may not be a need to do an expiratory hold to get this value. That said, the calculations may be less accurate with concurrent patient effort.[186]

If there is AutoPEEP, this means that something is getting in the way of the patient exhaling all the way back to baseline before a subsequent breath is delivered. This could be due to patient discomfort or need for more minute volume, but it can also be due to obstructive processes that get in the way of effective exhalation (i.e., asthma and COPD) or even inadvertent triggering of breaths. The fix on the vent interface would be to shorten our inspiratory time or decrease rate to lengthen the expiratory time (decrease the I:E ratio) and allow more exhalation; otherwise we could consider more sedation/ pain control and make sure we aren't accidentally triggering.[187] There is also the idea of utilizing applied PEEP to mitigate AutoPEEP and we'll touch on this idea later when we talk about an **Obstruction** strategy.[188]

One other thing we can do to eliminate AutoPEEP and reestablish our baseline at actual PEEP is to disconnect the patient from the vent circuit to allow a full and complete exhalation.[189] This is one of those rare cases in which it is OK to disconnect the vent circuit from the patient during transport for therapeutic reasons. Simply allow the patient to exhale and then reattach the circuit (and most likely canceling out a bunch of **Alarms** in the meantime). Just to make sure we understand how this works, let's draw it out as a waveform over time and label things along the way:



Just know that this is a temporary maneuver and that it is very likely the AutoPEEP will redevelop/ return if we simply resume previous vent settings. OK to use this technique to buy a bit of time, but it is not a solution, in and of itself.

To summarize, AutoPEEP is a movement of the pressure baseline above whatever we have dialed in for PEEP. While a small amount of AutoPEEP may be tolerable, its presence is always an abnormal finding. Issues with this are increased pressures (volume control) or decreased volumes (both volume control and

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[185] And more on this in **Vent Graphics** later

[186] Laghi & Goyal, 2012 - Very nerdy article on AutoPEEP in general, but specifically addresses all the various different ways for measuring it on the vent

[187] See sections on **Comfort**, **Triggers**, **Inspiratory Time (and I:E Ratio)**, **Types of Breaths**, and **Obstruction** for more on these things

[188] Stather & Stewart, 2005 - And for a preview of this idea (utilizing PEEP to mitigate AutoPEEP), refer to this article

[189] Do note that there are some patients in which disconnecting the circuit probably isn't the best option, this is mainly a concern with **Lung Injury** when we are particularly concerned with recruitment of alveoli

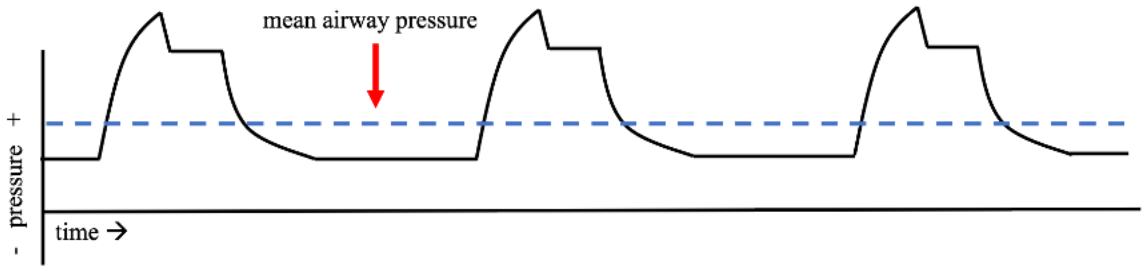
pressure control). Causes would be the inability to exhale fully, discomfort, and inadvertent triggering. Fixes include lengthening the expiratory time (decreasing I:E ratio), treating discomfort, avoiding accidental triggers, and utilizing applied PEEP.[190] In addition, we can reset AutoPEEP back to zero by temporarily disconnecting the vent circuit.[191]

Mean Airway Pressure

Last pressure to talk about for now is mean airway pressure. It's typically represented as P_{aw} (stands for airway pressure), sometimes as Pmean (mean pressure), and less often as MAP (mean airway pressure).[192] Mean airway pressure is the average pressure in the system throughout the respiratory cycle. There are formulas to estimate mean airway pressure, but it's probably easiest to just read off of our machine (assuming it's there).[193] We don't often use this pressure to guide treatment, but if we notice changes in mean airway pressure we can then look into details as to what changed in the system. For example, a high mean airway pressure can result from all sorts of things, each of which is a totally different issue: an increase in either **Peak Inspiratory Pressure** or **Plateau Pressure**, the presence of **AutoPEEP**, and increased **Respiratory Rate**. And same thing on the opposite end, lots of things can cause mean airway pressure to drop and we then must work to identify a specific cause.

One other thing about mean airway pressure is that it is strongly correlated with **Oxygenation**, particularly due to the variables of **Positive End-Expiratory Pressure** and **Inspiratory Time**.[194] More of either of these things leads to a higher mean airway pressure, so it can help to think of oxygenation in terms of this pressure and **Fraction of Inspired Oxygen**. Just recognize that too much of this good thing can turn bad - there is some evidence that an increased mean airway pressure is fairly well correlated with decreased hemodynamic function.[195] And while we commonly separate oxygenation into multiple concepts (as we did previously), it may be worth keeping this in mind as we look for trends in patient presentation.

We can conceptualize mean airway pressure in this way:



[190] Also addressing **Asynchrony & Dyssynchrony** patterns, it's just that this concept has not yet been discussed

[191] We sometimes see reference to the idea of both disconnecting the circuit and manually applying pressure to the patient's chest to facilitate full exhalation and the eliminate AutoPEEP, but it is not clear that this physical maneuver is supported by data and simply disconnecting the circuit will generally suffice

[192] And P_{aw} sometimes is used for airway pressure in general (i.e., the y-axis in the graphic on this page is airway pressure and that value changes throughout the breath), but we'll stick with it as a symbol for mean airway pressure in this manual

[193] We use one of these formulas to estimate mean airway pressure in the **Appendix** in order to demonstrate something about a **Hypotension** strategy we will outline later

[194] Lodeserto, 2018 - This page provides an alternative explanation for this relationship between mean airway pressure and oxygenation

[195] Su & friends, 2021 - This is similar to how **Positive End-Expiratory Pressure** decreases blood return to the heart

A General Vent Strategy

In this section we are going to summarize general parameters in each type of ventilation (i.e., each combination of mode and control) in order to demonstrate what settings and goals are shared among all methods and which are specific to certain types of ventilation. This general strategy is similar to what is often described as a lung-protective strategy that first came on the scene in regard to management of patients with ARDS. We've opted to present the two as distinct strategies and we'll come back to this idea when we get there.[196] We will also hash out a few of the differences in determining general settings for adults versus pediatrics. Just know that we go into much more detail on pediatric settings in a later section, this here is just the basics of the thing.[197]

Let's start with a discussion of things that apply to most ventilated patients, regardless of mode or control:[198]

$$TV = 6 – 8ml/kg\ IBW$$
$$MV \approx 100ml/kg\ IBW\ /min$$

if we choose a tidal volume of 6ml/kg and our goal minute volume is 100ml/kg IBW/min, then our calculated respiratory rate is 17:

$$MV = RR \times TV$$
$$100ml/kg\ IBW/min = RR \times 6ml/kg$$
$$100\cancel{ml/kg\ IBW}/min \div 6\cancel{ml/kg} = RR$$
$$17 \approx RR$$

Likewise, if we go with 8ml/kg our initial rate (to match that minute volume goal) comes to 13 per minute. Although it's not uncommon to see recommendations for an initial rate of 10 to 12 with adults, calculating a rate based on a minute volume goal is the preferred strategy. There are often good reasons to use a lower rate, but we'll get to those later.[199]

Moving forward, if we have a range of tidal volume to choose from, sometimes it just makes life easier to pick a nice, even number. For example, with an 80kg patient we end up with a tidal volume goal range of 480-640ml and a minute volume goal of 8L; it's totally fine to choose 500 or 600 or any value in that range. Just recognize that if we pick a higher value for tidal volume, we want a lower value for respiratory rate just to keep our minute volume approximately the same. This does not have to be exact, as we will adjust these settings as we go and work towards our goals moving forward. So we may choose a tidal volume of 500 and a rate of 16 (for a calculated minute volume of 8L). Or a tidal volume of 600 and a rate of 14 (for a calculated minute volume of 8.4L). Either is OK for now and we'll hone in our settings once we see how the patient responds to it all.

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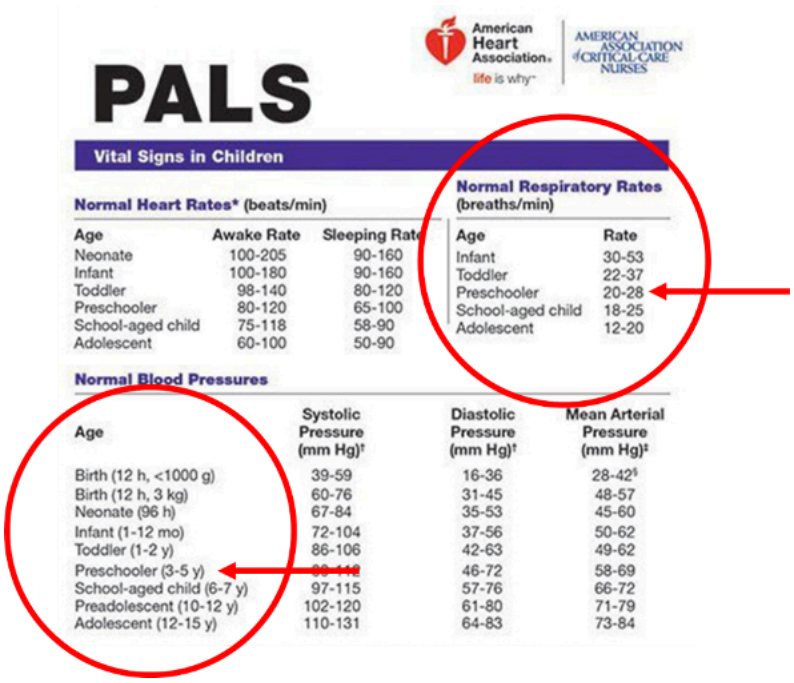
[196] We discuss the management of ARDS in the section on **Lung Injury**

[197] See **More on Pediatric Ventilation**

[198] Note that some patients do require different goals and we will discuss those shortly in **Specific Vent Strategies**; also, refer back to sections on **Tidal Volume**, **Ideal Body Weight**, **Respiratory Rate**, and **Minute Volume** for a discussion of these suggestions

[199] Specific examples of when we'd want to decrease rate below a normal range would be **Hypotension** and **Obstruction**

As for kids, the approach is to choose a rate in line with a reference card and disregard the above suggestion of 13-17/min. While this will result in an overestimation of minute volume, we can titrate values to address that later on.[200] For example, let's assume a 4-year-old child of 18kg. Based on this chart (again, from PALS) we want a rate in the 20-28/min range:[201]



PALS

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)‡
Birth (12 h, <1000 g)	39-59	16-36	28-42§
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	[illegible]	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

We can also use this chart based on the PALS data:[202]

age description	age (years)	rate	I-time (s)
infant	0.083 (1 month) - 1	30 - 53	0.3 – 0.7
toddler	1 - 2	22 - 37	0.4 – 0.9
preschooler	3 - 5	20 - 28	0.5 – 0.9
school-aged child	6 - 7	18 - 25	0.6 – 1.1
big kid	8 - 9	17 - 25	0.6 – 1.2
preadolescent	10 - 12	14 - 23	0.7 – 1.4
adolescent	12 - 15	12 - 20	0.8 – 1.7
adult	16 and up	12 - 20	0.8 – 1.7

[200] This overestimation of minute volume is because we use a higher respiratory rate for pediatrics, but still utilize a weight-based tidal volume (or tidal volume goal in pressure control); we refine this idea a bit in **More on Pediatric Ventilation**

[201] American Heart Association, 2016 (image) - There is a new chart of this same style in the 2025 PALS content, but all the values are unchanged/ the same as shown here

[202] As we mentioned before, see **Appendix** for an explanation of how we made this chart for normal respiratory rates and inspiratory times by age

And let's take these values and do a few calculations as so:

TV = 6 – 8ml/kg IBW
TV = 6 – 8ml/kg x 18kg
TV = 108 – 144ml

MV goal = 100ml/kg IBW/min
MV goal = 1800ml/min
MV goal = 1.8L/min

MV calculated = RR x TV
MV calculated = (20 – 28)/min x (108 – 144)ml
MV calculated = 2160 – 4032ml/min
MV calculated = 2.2 – 4L/min

The result here is a minute volume goal that differs pretty significantly from the calculated minute volume, but what to do with this information? We definitely want a minute volume (preferably measured as exhaled minute volume) that exceeds our normal quantitative goal of 100ml/kg IBW/min and also gives us an $EtCO_2$ in the normal 35-45 range, but let's start with 6-8ml/kg and a rate based on age and see how $EtCO_2$ looks after a few moments. This overestimation is particularly important, and maybe even lifesaving, if we decide to ventilate a child in volume control. There is always some dead space that we introduce into the system and this overestimation will help to mitigate that. Along those same lines, it may also be worth using a tidal volume on the higher end of the range (again, this is only for kids in volume control ventilation) to further mitigate this effect.[203] Last thing to note here is that we can, in fact, use weight-based minute volume goals for pediatrics that are specific to age groups and differ from the 100ml/ kg IBW/ min used for adults, but that discussion is deferred until **More on Pediatric Ventilation** - for now, this approach is just fine.

So we have tidal volume and respiratory rate all sorted, both for adults and kids. Next we need to consider the other parameters that are constant between modes and control methods. Let's put it into a chart just to make it easier to visualize. And this chart is basically a reflection of **Vent Parameters, Round One** with specifics added in:[204]

parameter	value	pro tips
TV	6-8ml/kg IBW	pick an easy number to work with that falls in that range
MV	adult: 100ml/kg IBW/min kiddos: TV x RR (&/or use a chart)	for adults, just take IBW in kg and move the decimal over (75kg IBW = 7.5L MV goal)
RR	adults: 13-17/min kiddos: use a chart	for kiddos: carry a reference card or have an app on a device to quickly reference pediatric values
FiO_2	1.0, then titrate down	we can titrate down in big jumps also, no need to go in small increments unless we have a good reason to do so
PEEP	5-6cmH_2O	for most vents this will be whatever the machine defaults to
I-time	adults: 0.8-1.7s kiddos: use a chart	normal for the average adult is 1.0s

Next step is to look at what extra parameters need to be dialed in on the machine depending on which mode and which control we choose for our patient. As said before, we can ventilate almost any patient in any mode and via any method of control, so long as we know what to monitor for depending on what we choose.

[203] For more on this idea of why we might want to use a tidal volume towards the higher end of normal for pediatrics, refer to the **Appendix**

[204] Weingart, 2010; Lodeserto, 2018 - Both recommend starting at an FiO_2 of 100% and then dropping down to 40 or 60% to see how the patient does; we can always titrate back up if need be, but if all is well we just leave it there (or even keep titrating down)

And if we are ventilating a patient in pressure control or SIMV (with pressure support), it's OK to just start with the defaults on whatever machine we are working with and then titrate from there given we do so in a timely fashion and with our ventilation goals in mind. Let's draw it all out in another chart: [205] [206]

	additional parameters
Assist Control Volume Control	none
SIMV Volume Control	***pressure support*** – start at 5-10cmH_2O and titrate as needed
Assist Control Pressure Control	***pressure control*** – start at 10-15cmH_2O and titrate to tidal volume goal
SIMV Pressure Control	***pressure control*** – start at 10-15cmH_2O and titrate to tidal volume goal ***pressure support*** – start at 5-10cmH_2O and titrate as needed
Assist Control PRVC	***pressure cap*** – set at 25-30cmH_2O (often by setting the high-pressure limit at 5cmH_2O above what we want this to be)
SIMV PRVC	***pressure cap*** – set at 25-30cmH_2O (often by setting the high-pressure limit at 5cmH_2O above what we want this to be) ***pressure support*** – start at 5-10cmH_2O and titrate as needed

[205] It's a bit tough to identify specific starting points for both pressure control and pressure support in the literature and recommendations vary a lot, but these are points to start off at and then we should always titrate towards exhaled tidal volume and exhaled minute volume goals as soon as possible; as for more insight into these initial settings:
Ashworth & friends, 2018 - They say start with pressure control at 5-10cmH_2O and limit driving pressure (plateau pressure or pressure control minus PEEP, which we will discuss later on **Driving Pressure**) to 16cmH_2O (which correlates with an additive pressure control of that amount)
Kneyber & friends, 2017 - These guys recommend limiting a driving pressure to 10cmH_2O for all (pediatric) patient types
Nagler & Chiefetz, 2019 - This duo suggests a starting pressure support of 5-10cmH_2O for kids
And just to be clear, all the pressures listed here (for pressure control and pressure support) are additive, not cumulative (and for a refresher on what that means, head back to **Types of Breaths**)

[206] Recall that pressure cap is a made-up term and is typically represented by 5cmH_2O less that what we set as the high-pressure limit

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At the expense of being overly redundant, let's combine the last two charts into another one to summarize how we determine vent settings, in general and for the normal patient: [207]

step one: **set &/ or calculate**		**step two:** **make a choice and dial in extra stuff**	
TV	6-8ml/kg IBW	**Assist Control** **Volume Control**	none
MV	adult: 100ml/kg IBW/min kiddos: TV x RR (&/or use a chart)	**SIMV** **Volume Control**	***pressure support*** – start at 5-10cmH_2O and titrate as needed
RR	adults: 13-17/min kiddos: use a chart	**Assist Control** **Pressure Control**	***pressure control*** – start at 10-15cmH_2O and titrate to tidal volume goal
FiO_2	1.0, then titrate down	**SIMV** **Pressure Control**	***pressure control*** – start at 10-15cmH_2O and titrate to tidal volume goal ***pressure support*** – start at 5-10cmH_2O and titrate as needed
PEEP	5-6cmH_2O	**Assist Control** **PRVC**	***pressure cap*** – set at 25-30cmH_2O (often by setting the high-pressure limit at 5cmH_2O above what we want this to be)
I-time	adults: 0.8-1.7s kiddos: use a chart	**SIMV** **PRVC**	***pressure cap*** – set at 25-30cmH_2O (often by setting the high-pressure limit at 5cmH_2O above what we want this to be) ***pressure support*** – start at 5-10cmH_2O and titrate as needed

In the ideal world, that's how we get vent settings for a specific patient. In the actual world we have a few things to consider (and we'll frame them as questions): What pathophysiologic changes affect the way this patient should be ventilated? What do we do with a patient already being ventilated if settings don't match what we come up with? How does this individual's body respond to all our theoretical stuff? The next few sections will answer these questions in turn. We will first look at specific situations that warrant alterations to this framework, then we will talk about setting up the vent in any scenario, and after that we will consider how to evaluate an individual's response to what we are doing with the machine and how we might adjust things to make him or her as happy as possible.

[207] In pressure control we don't actually set tidal volume, but we do need to have a value in mind and calculated out so that we can use it as a goal

Specific Vent Strategies

We have a chart from the last section that summarizes the initial calculations and choices we need to make for the average patient and depending on which types of breaths we want to deliver. Next step is to look at exceptions to the norm. To say it another way, sometimes a patient needs his or her breaths delivered in a specific way (different to what we identified as normal) due to intricacies of a given illness or disease or physiologic state. We sometimes take those normal parameters and alter them to meet specific needs and issues. It's totally OK to break the rules we've established so far, as long as we know when and how to do it and can justify a good reason. We will look at a few situations, in turn, to see how it all looks.

Vent strategies are often presented as a choice of two distinct categories: the injured or sick lung approach and an obstructive strategy.[208] We've opted to present this decision-making process as a set of five possible strategies from which providers can choose. This approach is specific to the emergency setting where preventing acute decompensation is a primary concern. First is the general strategy discussed just now, the other four include obstruction, hypotension, acidosis, and lung injury. There is no right or wrong in this process (i.e., it's often just fine to use a two-strategy approach), we just think it makes sense to take things a bit further as we have outlined in the following sections.

To elaborate on this idea for a moment: these four alternate strategies presented in the following sections are aggressive ventilator strategies that we use to avoid acute decompensation in patients who present with specific pathophysiologic patterns. It is not the case that all hypotensive patients need a tailored approach (i.e., we can often manage that via pharmacologics) or that all acidotic patients are at risk of hemodynamic collapse (i.e., many are stable and not at risk for acute deterioration), but it is important to know these patterns in order to implement them when indicated. Understanding these approaches and the mechanisms behind them will allow us to quickly adjust our ventilator approach when time is of the essence. And then it will often be the case that we can subsequently revert towards the general strategy when the acute phase has passed.

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[208] To provide more context on this two-strategy approach:
The ARDS Network, 2000 - This was a major paper that led the movement towards lower tidal volume with ventilated patients; while it focuses on a specific patient group (i.e., that injured lung cohort), it set the stage for further research into the idea of much lower tidal volumes than were initially used
Weingart, 2010; Weingart, 2016b - A podcast series and paper, respectively and by the same human, that outline this two-strategy approach to vent management; while directed towards emergency department physicians, the content is 100% applicable to those of us that work in the transport setting

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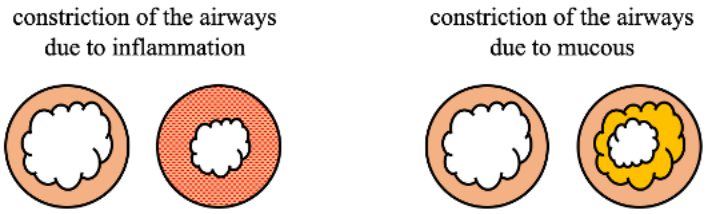
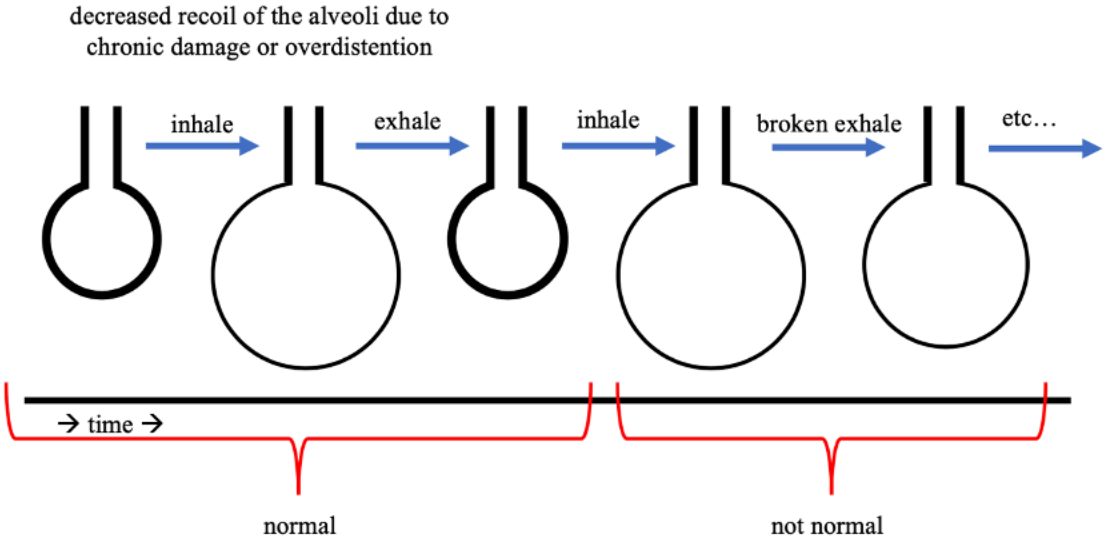
Obstruction

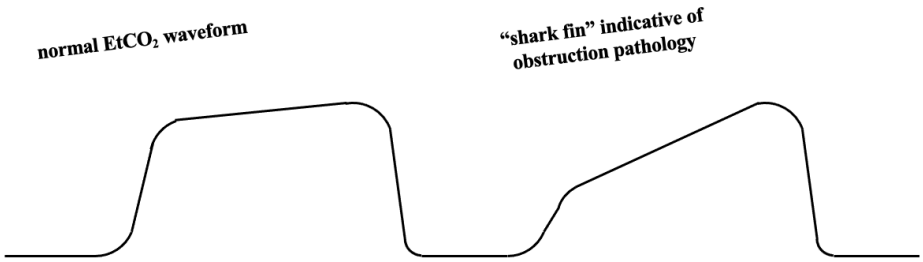
In patients with symptoms of acute asthma, COPD, and/ or allergic reaction, there is often a problem of breath stacking or **AutoPEEP** because the patient is unable to exhale fully in a normal amount of time. The pathophysiology is multifaceted and varies a bit depending on underlying cause, but often includes some sort of narrowing of the airways:



And also decreased recoil of the alveoli which results in inadequate exhale or an exhale that requires either forced effort (by the patient) or more time:



Evidence for this sort of pathophysiologic process is most likely clinical (i.e., based on physical exam and patient history), but can also be inferred from the presence of **AutoPEEP** (measured via an expiratory hold or observed on **Vent Graphics**) or maybe with a specific "shark-fin" pattern on $EtCO_2$:[209]



[209] Just note that the "shark-fin" is not always seen and that the absence of it does not imply that no obstructive pathology is present

The fix is to adjust vent parameters to allow for more time at exhalation. We do this by extending or lengthening the expiratory time (decreasing I:E ratio). A normal I:E ratio is 1:2-3 and we can decrease that by decreasing either the **Inspiratory Time** or **Respiratory Rate**.[210] A good starting point in this population is an I:E ratio of around 1:5. The typical way to get here is to decrease rate (and also inspiratory time) until we see an I:E ratio in that range that we want. The machine normally does this calculation for us, but just as an example we'll show it all here:

with I-time 1.0s and RR 17:
60 ÷ 17 breaths ≈ 3.5s/breath
3.5s – 1.0s (I-time) = 2.5s
∴ I:E ratio = 1:2.5

with I-time 1.0s and RR 13:
60 ÷ 13 breaths ≈ 4.6s/breath
4.6s – 1.0s (I-time) = 3.6s
∴ I:E ratio = 1:3.6

with I-time 0.8s and RR 13:
60 ÷ 13 breaths ≈ 4.6s/breath
4.6s – 0.8s (I-time) = 3.8s
∴ I:E ratio = $\frac{0.8}{0.8}$: $\frac{3.8}{0.8}$
I:E ratio = 1:4.8

So even if we drop both respiratory rate and inspiratory time to the lower ends of normal parameters, we end up with an I:E ratio shy of what we want for these obstructed patients. Let's keep up with some of these calculations and put them all side by side:

I-time 1.0s		**I-time 0.8s**	
rate	***I:E ratio***	***rate***	***I:E ratio***
17	1:2.5	17	1:3.4
16	1:2.8	16	1:3.7
15	1:3.0	15	1:4.0
14	1:3.3	14	1:4.4
13	1:3.6	13	1:4.8
12	1:4.0	12	1:5.3
11	1:4.5	11	1:5.8
10	1:5.0	10	1:6.5

[210] And as noted back in **Inspiratory Time (and I:E Ratio)**, normal is 1:2 and the 1:3 is more due to convention that what the patient would breathe at if left alone to nature

Now let's assume we choose an inspiratory time of 1.0s and a rate of 10 (for a calculated I:E of 1:5.0), what does that do to our other parameters? Biggest thing that will be affected is **Minute Volume**. We'll do some calculations to demonstrate the impact on a 65kg **Ideal Body Weight** patient with a **Tidal Volume** of 8ml/kg:

MV goal = 100ml/kg IBW/min
MV goal = 100ml/kg IBW/min x 65kg
MV goal = 6500ml/min
MV goal = 6.5L/min

TV = 8ml/kg x 65kg
TV = 520ml

MV calculated = TV x RR
MV calculated = 520ml x 10/min
MV calculated = 5200ml/min
MV calculated = 5.2L/min

In fact, we'd have to go up to a tidal volume of 10ml/kg to get to our minute volume goal:

MV goal = TV x RR
6500ml/min = TV x 10/min
TV = 650ml

65kg x ?ml/kg = 650ml
? = 10ml/kg

Now the priority in this strategy for the obstructed patient is to avoid AutoPEEP and allow full exhalation, so start with a focus on rate and I:E ratio. If we need a starting point for tidal volume, just go with the high end of normal or 8ml/kg. Then go down on rate to an I:E ratio of at least 1:5 and monitor for AutoPEEP. After that, we can then titrate up on tidal volume to maintain minute volume as possible. If for some reason we can't go too high on tidal volume due to a high plateau pressure and/ or we have a very low rate due to continued AutoPEEP, minute volume may end up below goal. That's OK in the short term, we just want to try and get as close to it as possible while still allowing for full exhalation and avoiding the AutoPEEP issue.[211] We will simultaneously be doing pharmacologic interventions (Albuterol, Magnesium Sulfate, Ketamine, Epinephrine, whatever our agency endorses) and hopefully the reason for this alternative strategy can get reversed to some degree and then we can go up on rate and work our way back to normal parameters.[212]

In **Pressure Control**, we still drop the rate (and maybe inspiratory time too) to lengthen expiratory time (decrease I:E ratio), but we also want as much volume per breath to try and get as close to our minute volume goal as possible. Instead of a pressure control of 10-15cmH_2O, consider going straight to the top and starting at 20-25cmH_2O to see what our exhaled tidal volume values look like.[213] If ventilation is above goal with this higher pressure control, we should then titrate down.

With these obstruction cases, it may be tempting to drop **Positive End-Expiratory Pressure** to zero to better allow the patient to exhale. The thought process goes like so: if they are breathing out while we are pushing air in, this has the potential to be problematic. That said, there is some thought that applied PEEP can

[211] Pruitt, 2007; Yartsev, 2023g - The first provides a more in-depth discussion of this permissive hypercapnia approach; the second gives way more information that we thought possible on the potential effects that such an approach may have (but of note, one of those effects may be bronchodilation)
[212] See **Medications in Mechanical Ventilation**, specifically **Medications for Obstruction**
[213] A pressure control value of 25cmH_2O gives us the upper limit for a safe plateau pressure, assuming a PEEP of 5cmH_2O and an additive pressure control value; that said, there is a good argument that **Driving Pressure** ought to be limited, so this is a bit of a grey area

help fix AutoPEEP, but we do want to keep applied PEEP lower than AutoPEEP. Just know that we may want to maintain PEEP at our minimum of 5cmH_2O to maximize **Oxygenation** and help recruit more alveoli, but sometimes we let that go in order to avoid AutoPEEP. There may be a happy middle ground with a PEEP somewhere between zero and a normal value, but there isn't much content on that and we'll leave it as a maybe in the overall scheme of things.[214]

And regarding resolution of the problem: if we have decreased our I:E ratio to accommodate exhalation and we end up at a point where AutoPEEP is consistently zero, we can then titrate our I:E ratio back to normal to make things more comfortable for the patient. This allows us to work back towards our minute volume goal that we started with, as it is likely that our minute volume will be below that goal with a much lower respiratory rate. If things change and obstruction recurs (and then we notice AutoPEEP all over again), we can go back to the smaller I:E ratio (i.e., longer expiratory time). The idea here is that we are constantly reassessing and making these small adjustments to optimize ventilation to the patient in a given moment. Just because a smaller I:E ratio was warranted at the start doesn't mean it is needed forever.

Another thing to mention that is relevant to some types of obstruction is that patients with chronic lung disease (i.e., COPD) may have a $PaCO_2$ (and therefore an $EtCO_2$) that is above a normal baseline. In these patients with chronic retention of carbon dioxide it can be detrimental to over-ventilate in an effort to normalize $EtCO_2$, therefore **Ventilation** should be goal-directed using a weight-based goal.[215] If that goal is met and AutoPEEP is minimized, it is OK that $EtCO_2$ remains high. And again, this is not for all obstructive pathologies, just in the case of COPD (i.e., a normal $EtCO_2$ target may be just fine with asthma and/ or allergic reaction).

One more confounding factor here is that both PEEP (including AutoPEEP) and a high FiO_2 (i.e., oxygen administration) can lead to an increased gap between $PaCO_2$ and $EtCO_2$.[216] This means that $EtCO_2$ may be a less reliable metric in most ventilated obstruction patients, not just those with COPD.[217] That said, it still makes sense to use it as a tool and trend changes as we do with all patients, but recognize that an elevated $EtCO_2$ paired with an adequate weight-based minute volume is likely OK.

To summarize our obstruction strategy: utilize a lower respiratory rate (and consider a shorter inspiratory time also) to a goal I:E ratio of 1:≥5. Consequently, we need to titrate tidal volume (or pressure control) up as far as the patient's lungs will allow.[218] Know that we will likely be short on our minute volume goal and that's OK. As our pharmacologic interventions start to work we can hopefully migrate back towards normal parameters to meet ventilation goals. Maybe consider adjusting PEEP, but know that there isn't yet a good consensus on that. Also, be sure to check for AutoPEEP periodically and consider disconnecting the vent circuit to reset it back to zero if need be.[219]

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[214] Stather & Stewart, 2005 - In addition to exploring this idea of using PEEP to mitigate AutoPEEP, these two also provide an overview of a strategy for the asthmatic patient in general

[215] Farkas, 2024a - More on this idea in this free e-book of sorts from EmCrit

[216] Yartsev, 2025a - We cited this page earlier when we discussed $EtCO_2$ as an indicator of **Ventilation**

[217] Kartal & friends, 2011 - Older paper that looks at sidestream $EtCO_2$ analysis in COPD, demonstrates poor correlation at $PaCO_2$ in this patient population

[218] Remember that it may be harder to get complete exhalation in pressure control ventilation (versus volume control) due to differences in how those breaths are delivered (i.e., decelerating flow versus constant flow, see **Types of Breaths** to review this idea)

[219] We discussed this technique of disconnecting the vent circuit for therapeutic reasons in the section on **AutoPEEP**

Hypotension

In patients with hypotension the primary concern is that mechanical ventilation can decrease cardiac output and further contribute to the problem. We discussed this already in reference to positive-pressure ventilation generally and both **Positive End-Expiratory Pressure** and **Mean Airway Pressure** specifically.[220] We mentioned then that fluid volume seems to mitigate this effect, so first strategy here (since we are committed to positive-pressure ventilation) is to replace fluids/ resuscitate if not contraindicated. Next step is to restrict that PEEP to whatever minimum value we need to maintain adequate oxygenation. Beyond that, we can limit the time spent at inspiration during the overall respiratory cycle. Think of it this way: preload drops further when we increase intrathoracic pressure, so if we decrease the amount of time spent pushing air into the system we can limit this effect.

It is worth noting that most clinicians who use ventilators also have access to vasoactive medications that can be used to treat or avoid hypotension. In these instances or clinical settings the more common approach is to address refractory hypotension pharmacologically and simply mitigate the effects that mechanical ventilation has on blood pressure by titrating those medications.[221] That is completely acceptable, but recognize that those tools may not always be available (i.e., a remote or austere setting) and there may also be a case for doing both simultaneously (i.e., profound hypotension or peri-arrest state).

Now to quantify the idea of how blood return and cardiac output are affected due to breaths given by the machine, consider two patients: one at a **Respiratory Rate** of 17 and one at a rate of 10. If we assume an **Inspiratory Time** of 1.0s (norm for the adult patient), let's calculate how much time the patient experiences a state of decreased preload (i.e., inspiration). We've labeled this idea as percentage of time at decreased preload (%TaDP):

$$\% \text{ TaDP} = (\text{RR} \times \text{I-time}) \div 60\text{s}$$

with RR of 17

$$\%\text{TaDP} = (17 \times 1.0\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 17\text{s} \div 60\text{s}$$
$$\%\text{TaDP} = 28\%$$

with RR of 10

$$\%\text{TaDP} = (10 \times 1.0\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 10\text{s} \div 60\text{s}$$
$$\%\text{TaDP} \approx 17\%$$

We can further drop this percentage by decreasing inspiratory time:

with RR of 10 and I-time 0.8s

$$\%\text{TaDP} = (10 \times 0.8\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 8\text{s} \div 60\text{s}$$
$$\%\text{TaDP} \approx 13\%$$

[220] See **How is Positive-Pressure Ventilation Different?** to review our discussion on the negative effects of positive-pressure ventilation

[221] And see **Therapeutics for Hypotension** in the chapter **Medications in Mechanical Ventilation** for a discussion of this

By dropping respiratory rate to 10 (from 17) and decreasing inspiratory time to 0.8s (low of normal for the adult patient), we can cut the amount of time at decreased preload by over half. While we could keep dropping respiratory rate, we stop at 10 because we need to maintain minute volume in these patients. Let's look at what happens to **Minute Volume** if we drop the rate to 10 and then come up with a strategy to address it. As before, we'll assume a patient with an **Ideal Body Weight** of 65kg and a **Tidal Volume** of 8ml/kg:

MV goal = 100ml/kg IBW/min
MV goal = 100ml/kg IBW/min x 65kg
MV goal = 6500ml
MV goal = 6.5L/min

TV = 8ml/kg x 65kg
TV = 520ml
MV calculated = TV x RR
MV calculated = 520ml x 10/min
MV calculated = 5200ml/min
MV calculated = 5.2L/min

Now 5.2L/min isn't super far off from 6.5L/min, but we need to remember that a hypotensive patient is likely at risk of shock and, therefore, we need to make sure that **Ventilation** is adequate by delivering at least what our calculated minute volume minimum is.[222] This idea is in stark contrast to the obstruction strategy in which we decided it was OK to let minute volume fall below goal; in hypotension we need to maintain (or even exceed, especially with **Acidosis** – discussion on that to follow) our minute volume goal. So let's titrate tidal volume up to 10ml/kg and see where we end up:

TV = 10ml/kg x 65kg
TV = 650ml

MV calculated = TV x RR
MV calculated = 650ml x 10/min
MV calculated = 6500ml/min
MV calculated = 6.5L/min

If we drop respiratory rate to 10 (and inspiratory time to low of normal by age) to minimize the percentage of time spent at decreased preload (i.e., inspiration) and increase tidal volume to 10ml/kg, then we maintain our minute volume goal of 100ml/kg IBW/min. Now that we've logically arrived at a strategy of decreased rate and increased tidal volume, let's rewrite the order of the steps as so: increase tidal volume first, then decrease respiratory rate to match minute volume goal. The reason for this is that we don't want to arbitrarily drop respiratory rate and then wind up in a situation where we can't titrate tidal volume up to goal. That would result in a decreased minute volume (which we said is an important thing in the patient at risk for shock). So let's go up on tidal volume as much as we can (even beyond 10ml/kg if we can maintain a safe **Plateau Pressure**) and then drop rate afterward. Even if we aren't able to drop the percentage of time at decreased preload (%TaDP) by half as in the example shown, we can at least move in that direction while ensuring adequate ventilation.[223]

Now there are other justifications for using a high tidal volume and low respiratory rate strategy that don't include this percentage of time at decreased preload (%TaDP) concept, we just find that this makes it easy

[222] Mannarino, 2014 - Refer to this video for a review of what shock is and how it is related to oxygen delivery
[223] Another advantage of titrating tidal volume first and then rate is that it allows the strategy to be applicable to both adult and pediatric patients without having to come up with more age-based recommendations; while this may or may not be a good reason in and of itself, it does help to keep processes simple and applicable across the board…

to appreciate. An alternative justification would be that the strategy decreases dead space.[224] We talked about this idea back when we discussed making changes to address minute volume needs and the idea is that dead space gets introduced with each breath given, so fewer breaths (with more volume each) means less dead space overall.[225] Another rationale would be **Mean Airway Pressure**. This high tidal volume, low respiratory rate approach decreases average pressure into the system, especially when we consider lowering PEEP towards zero (i.e., using the bare minimum necessary to maintain oxygenation). While lowering mean airway pressure could negatively impact **Oxygenation**, we may be able to counteract that with higher FiO_2 to meet our goals. The point here is that there are multiple justifications for this strategy; one has been spelled out here and the other two are deferred until the **Appendix**.

Another point to make here is that not all hypotensive patients are hypotensive for the same underlying, pathophysiologic reasons. As a general rule, decreasing %TaDP or mean airway pressure is helpful, but there is a subset of patients who present with hypotension specifically due to left-ventricular failure - in these cases, PEEP (and subsequently a higher mean airway pressure) may actually help to improve cardiac output. A detailed discussion of this concept is deferred until **Therapeutics for Hypotension**.

One last thing about this hypotensive strategy. Similarly to the **Obstruction** approach, if we arrive at a point where we've mitigated the risk related to hypotension with adequate resuscitation and/ or other interventions (such as vasoactive medications) we can always work back towards normal settings. Even though we put an upper limit on tidal volume by monitoring plateau pressure, recognize that lower values are still preferred for alveolar safety. We'll touch on this again when we get to **Titrating Up on Tidal Volume?**, but realize that if this hypotension strategy is a temporary solution that may not need to be sustained for the entirety of transport or patient interaction.

To summarize: in the hypotensive patient we want to decrease the amount of time spent at decreased preload (%TaDP) while maintaining minute volume at our weight-based minimum. To do this, we drop inspiratory time to low of normal, increase tidal volume as much as we safely can manage (in pressure control this may mean starting at 20-25cmH_2O), and then decrease rate while monitoring minute volume. We also want to be cautious of high PEEP while recognizing that oxygenation (facilitated by PEEP) is important in these patients with potential low perfusion states. Said one more time in the short and sweet manner of things: when ventilating the hypotensive patient, drop inspiratory time, increase tidal volume, drop rate (to maintain minute volume goal), and keep PEEP to a minimum.

[224] Bauer, 2015 - While the strategy discussed in this podcast is slightly different than ours (and includes decreasing PEEP all the way to zero), the basic idea is the same

[225] We discussed this idea of how dead space changes with comparable titrations to respiratory rate and tidal volume back in **Ventilation**

Acidosis

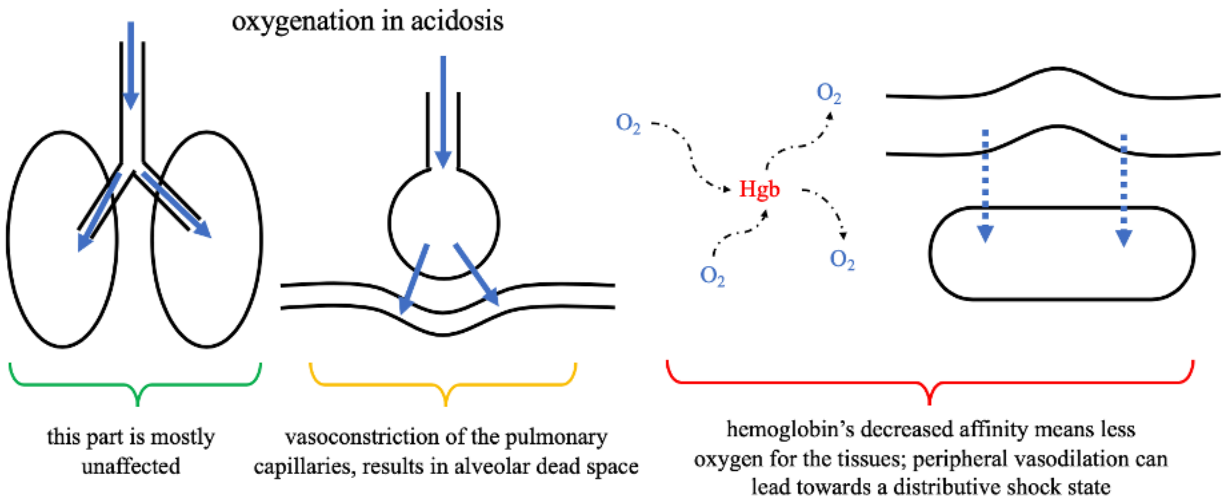
Acidosis is the buildup of hydrogen ions in the blood.[226] It can be due to many things: inadequate tissue perfusion (i.e., shock states), metabolic processes or pathologies (i.e., diabetic ketoacidosis, loss of bicarbonate), respiratory failure (i.e., opiate overdose), ingestion of exogenous substances (i.e., all the causes of elevated anion gap metabolic acidosis), etc. When hydrogen ion concentration in the blood goes up and therefore pH goes down, there are a number of negative consequences that can affect homeostasis: cardiac function is compromised, electrolyte shifts occur, oxygen binding to hemoglobin is affected, vasoactive medications become less effective, etc. For all of these reasons, it is important to limit the progression of an acidotic state.

Now the body does have mechanisms for compensating against acidosis, both respiratory and metabolic. In a state of respiratory acidosis, the body reacts metabolically by increasing the concentration of bicarbonate to keep pH in a normal range. In a state of metabolic acidosis, the body compensates via the respiratory system by increasing **Minute Volume** in order to exhale more carbon dioxide; again, in an effort to keep pH in a normal range. This is where we can facilitate the process using mechanical ventilation - by increasing, or maintaining an increase in, minute ventilation.

All that said, the need to address an acidotic state with mechanical ventilation is more relevant when the state of acidosis is markedly pronounced. Said another way: it probably isn't necessary to implement an acidosis vent strategy when the condition is borderline or minor and can be managed by other means, but it is warranted when the patient's acidosis is severe or when there is a risk to hemodynamic stability. As far as when to make that determination or what triggers exist for when an acidosis vent strategy should be implemented, there is no clear evidence and most that does exist is based on pH alone or an ABG, neither of which is routinely available in transport. That said, consider implementing the acidosis vent strategy if there is hemodynamic instability with concurrent or suspected acidosis. This could include hypotension, initial use of vasoactive medications, or increased need for vasopressors (i.e., increasing dose requirements); presence of new cardiac dysrhythmias and/ or ectopy; or signs of poor perfusion on assessment.

[226] And more on all of this in **Blood Gas Analysis**

As for the oxygenation component: we mentioned already that certain factors can impact hemoglobin's affinity for oxygen and pH is one of those things.[227] More acid and lower pH means that oxygen is held less tightly by hemoglobin as it moves through the blood (i.e., hemoglobin's affinity for oxygen is decreased). This makes it more difficult to get oxygen to the tissues where it is needed.[228] While we can increase the amount of oxygen to the alveoli and expect it to diffuse into the blood, getting it from that dissolved state to the hemoglobin itself is the challenge. And then when we do get it loaded onto hemoglobin, it may offload early before getting to the tissues where we intended it to go. To summarize these ideas, here's how we would draw it all out:[229]



While we still do all the things we already know how to do in regard to **Oxygenation**, the acidosis strategy outlined here helps to correct these shifts so that oxygen delivery returns to normal. This is in addition to preventing the progression of an acidotic state, perhaps even reversing the thing to some degree, by increasing or maintaining a high **Minute Volume**.

To expand on this, the classic example here is a patient in diabetic ketoacidosis breathing at a **Respiratory Rate** of 30: flight crew comes along, paralyzes and intubates the patient, and then sets the vent up at a "normal" rate of 12. The patient had been compensating with an increased respiratory rate, but that compensation gets taken away suddenly. As a result, the patient quickly decompensates, crashes, and suffers a less-than-ideal outcome.[230] We've already reviewed the ideas of both oxygenation and the fact that a rapid increase in $PaCO_2$ and concurrent drop in pH can lead to hemodynamic issues such as decreased cardiac contractility, lessened response to catecholamines, and systemic vasodilation.[231] Given all of this, we need a strategy that maintains minute volume both to prevent these hemodynamic effects and to ensure oxygen delivery to the tissues.

While a bit tricky to pinpoint exactly what our minute volume goal ought to be, let's start with a minimum goal double that of the normal patient: 200ml/kg IBW/min.[232] To achieve that goal, we likely need to increase both rate and **Tidal Volume**. In order to increase minute volume and get our $EtCO_2$ within a normal

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[227] This was back in the section on **Oxygenation**

[228] Hasudungan, 2018; Smith, 2014 - The first is a video that reviews this concept of a right shift and also covers some basic physiology we discussed before; the second is a quick video to review acid-base analysis (and for more resources on this, look forward to the section **Blood Gas Analysis**)

[229] Lumb & Slinger, 2015 - Refer back to this article for a review of **Hypoxic Pulmonary Vasoconstriction**

[230] Weingart, 2009 - And while we don't focus on the intubation process in this manual, look to this throwback episode of EMCrit for a strategy on how to do so while simultaneously avoiding the problems we discuss here

[231] Carter & friends, 2010 - This article focuses how oxygen delivery is affected by acid-base imbalances, but also goes into detail about the consequences of an increased $PaCO_2$ that we mention here

[232] Weingart, 2010 - Our suggestion vaguely resembles the one recommended here (double minute volume to drop carbon dioxide from 40 to 30, that's with a starting minute volume of 120ml/kg/min); that said, this 200ml/kg/min figure is a minimum starting point and we may need to take it further than that – the idea is that we initiate ventilation to prevent immediate deterioration and then go from there to work towards goals (as outlined later in this section)

range we typically start by changing tidal volume first and then respiratory rate.[233] The reason for this is that adding a breath also adds in dead space to the equation. In the acidosis situation, however, the patient is likely already breathing fast, so let's just use a high of normal tidal volume (i.e., 8ml/kg) and see what kind of rate we'd need to get to this increased minute volume goal of 200ml/kg IBW/min:

MV goal = 200ml/kg IBW/min
MV goal = 200ml/kg IBW/min x 65kg
MV goal = 13000ml/min
MV goal = 13L/min

TV = 8ml/kg x 65kg
TV = 520ml

MV goal = TV x RR
13L = 520ml x RR
13L/520ml = RR
25 = RR

This means that a tidal volume at 8ml/kg and a respiratory rate of about twice normal will get us the theoretical minute volume of 200ml/kg IBW/min. We could also potentially go higher than 8ml/kg, so long as we make an effort to ensure no injury is caused in the process.[234]

In the normal patient, this sort of increase in minute volume would drive our $EtCO_2$ down significantly and create a state of respiratory alkalosis, but we said already that this compensatory respiratory rate is what we want. Now we just need to figure out how to measure or quantify to what extent we are helping the patient. We mentioned in a footnote that this figure (the 200ml/kg IBW/min one) is just a starting point, we then need to be a little more exact moving forward. There are a few strategies and we'll talk about them stepwise in order of least exact to more exact.

First thing we could do is to match our rate on the vent to the rate at which the patient was breathing before we took that respiratory effort away. This assumes that the patient was compensating adequately beforehand and that we are the ones intubating or taking that airway away. It also assumes that the mechanical breaths we give will go in and out just as easily as they were when the patient was breathing spontaneously - this may not actually be true, however, due to the fact that there may be sedatives and/ or paralytics at play that inhibit the patient's forced expiration.[235] And then the last downside is that this approach doesn't give us a quantitative goal to work towards (i.e., something more specific than simply a large minute volume).

Another strategy would be to measure the patient's $EtCO_2$ prior to intubation. We can do this via a side-stream nasal cannula device or by using an in-line $EtCO_2$ device with non-invasive ventilation.[236] We can then increase minute volume via rate and/ or tidal volume and fine-tune to this $EtCO_2$ that the patient was at before we messed with things. This strategy is similar, in part, to the above strategy in that it requires that the patient was compensating adequately on his or her own before we intervened.

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[233] Deciding which parameter to change (tidal volume versus respiratory rate) to alter minute volume was discussed in **Ventilation**
[234] Discussed further in **Titrating Up on Tidal Volume?**
[235] Bishop, 2026 - And the solution, therefore, would be to monitor for **AutoPEEP** or consider increasing **Tidal Volume** instead; also see **Medications in Mechanical Ventilation** for a discussion on these different agents
[236] Weingart, 2009 - We referenced this podcast already on the previous page, but take a look for a step-by-step discussion of this strategy for intubating the acidotic patient while maintaining minute volume in the preoxygenation phase with non-invasive positive-pressure ventilation

A third approach is to utilize Winter's Formula to establish an $EtCO_2$ goal. The formula looks like so:

$$PaCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$$

The formula is designed to measure the respiratory component with a known metabolic acidosis (i.e., measured $PaCO_2$ is compared to a calculated $PaCO_2$ to determine if the patient is compensating adequately or if a mixed disorder is present),[237] but we can modify its use in the transport setting to guide our titration of $EtCO_2$ (via minute volume).[238] This strategy is of use if we are taking over care of an acidotic patient who is already on the ventilator:

$$EtCO_2 \text{ should be} \leq (1.5 \times HCO_3^-) + 8$$

If we use this modified formula, adjust minute volume to that goal, and get our $EtCO_2$ right at the calculated value based on HCO_3^- from labs, we still may be a bit shy of our minute volume goal. Just keep that in mind and know that's why we wrote it out as we did without the "±" and with the "≤." If it helps, think of this value we get from the modified Winter's Formula as an upper limit for $EtCO_2$ in the patient with metabolic acidosis. One more point to make is that the HCO_3^- can be from either the metabolic panel or blood gas for our use in the transport setting, but know that there are varying opinions on that.[239] Regardless of source, however, it is best if the HCO_3^- is from a current set of labs. The longer it has been since the value was initially obtained, the less applicable that value becomes to the current situation.

In the event that the most recent HCO_3^- is more than a few hours in the past and that no prior data exists on pre-intubation respiratory rate or $EtCO_2$, the situation becomes a little less clear. We can still aim for that minute volume goal of 200ml/kg IBW/min and use the Winter's Formula approach, especially in cases where the patient's ventilation has not been maintained at these goals since labs were measured. We may also consider decreasing sedation to allow the patient to better express a need for more minute volume and evaluate things subjectively; this also allows the patient to actively participate in exhalation which will shorten the total breath cycle time (i.e., allow for a higher rate with no complicating **AutoPEEP**). We could even make it easier for the patient to trigger breaths by titrating **Triggers**.

To bring it all home, we can do all of these strategies together: try to match the patient's respiratory rate and $EtCO_2$ as measured before intervention, then compare both minute volume to our calculated minimum goal of 200ml/kg IBW/min and $EtCO_2$ to a goal derived from Winter's Formula. And if we aren't the ones taking the airway and intubating, the Winter's Formula approach is likely the best strategy. If that isn't an option, however, we may be able to let the patient guide treatment by decreasing sedation and trigger thresholds and allowing the patient to take the lead. The only next best thing here would be to measure gases en route to see how the patient is responding to treatment, but most of us don't have that capability in the field and we'll withhold a discussion of it until the section on **Blood Gas Analysis**.

One more thing to mention in regard to modes: because we are shooting for a high minute volume in the acidotic patient, **Assist Control** is the better option for these patients. If we do go with **Synchronized Intermittent Mandatory Ventilation** and the patient has spontaneous effort to breathe, consider increasing pressure support so that patient-triggered breaths match machine-delivered ones (and this would avoid a drop in minute volume if we were following the normal SIMV strategy of pressure support breaths below tidal volume goal).[240] In either case, the idea is to match the patient's need for more minute volume as expressed by effort to trigger more breaths. As always, monitor airway pressures and be sure that **AutoPEEP** is not an issue. We

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[237] Foster & Grasso, 2014 - Short video to explains Winter's Formula and its normal use in a clinical setting

[238] Lodeserto, 2018 - See *Part 3* of this series, it gives another perspective on how to manage the ventilated patient with concurrent (severe) metabolic acidosis

[239] Nargis & friends, 2015 - This is because in the metabolic panel it is a measured quantity, in the arterial blood gas it is calculated and there can be some discrepancy between the two values; all that said, there is strong correlation between the two and it likely doesn't much matter in the majority of cases; and while this particular study was looking at the totally unrelated idea of cost-effectiveness related to blood gas analyzers in the developing world, the findings on correlation between the two values are still worthwhile

[240] We talked about this idea that pressure support breaths are often smaller than the time-triggered or mandatory ones way back in the section on **Synchronized Intermittent Mandatory Ventilation**; also review **Assist Control** mode

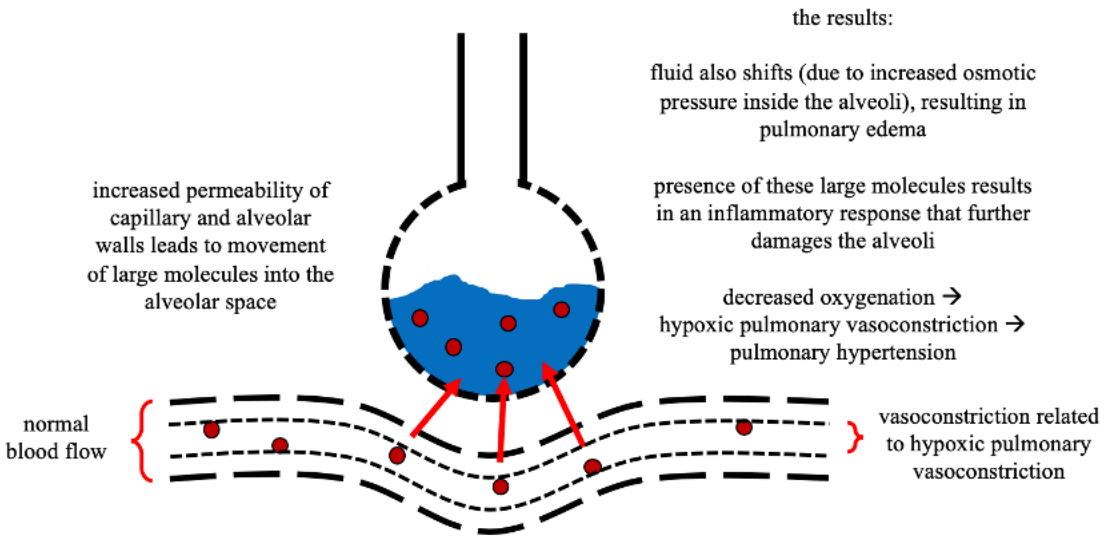
definitely don't want to get in a situation where minute volume drops due to the development of AutoPEEP and the cycling off of breaths due to our high-pressure alarm.[241]

We went on a bit of a tangent here, so let's get back to our vent strategy for the acidotic patient: use a tidal volume goal high of normal (8ml/kg, maybe even more) and increase rate (either to match patient's intrinsic rate, if possible, or even just double normal for the patient's age), then aim for a goal minute volume of 200ml/kg IBW/min and an $EtCO_2$ of patient's baseline prior to intervention or as determined by Winter's Formula. The goal of the acidosis strategy is to maintain minute volume in order to prevent oxygen delivery from getting worse, to work towards correcting the underlying imbalance, and to prevent any acute decompensation from a rapid rise in $PaCO_2$.

Lung Injury

Another much-discussed strategy in vent management is the injured or sick lung strategy, also known as the lung-protective approach. This is for patients that have lungs particularly susceptible to further injury or barotrauma and, as a result, we use less volume per breath in an effort to avoid overinflation. We then have to increase **Respiratory Rate** to maintain **Minute Volume** or be OK with an elevated $EtCO_2$. Another component of this strategy is higher than normal **Positive End-Expiratory Pressure**, which helps via a few different mechanisms. We'll start by reviewing the concept of acute lung injury and discussing the pathophysiology of acute respiratory distress syndrome, then we'll get into specifics about vent strategy.

Acute lung injury (ALI) refers to a number of pathologies that inhibit normal pulmonary gas exchange.[242] Specific causes include sepsis, pneumonia, bleeding from a traumatic injury, inhalation of toxins or smoke, and aspiration. Acute lung injury is a concept that lives on a spectrum with acute respiratory distress syndrome (ARDS) being the most severe endpoint. While acute lung injury, as a term, may also be described as mild or moderate ARDS, the underlying pathophysiology is the same. The main component of the disease process is that the alveolar and capillary walls become permeable to stuff that is normally sequestered in the blood:



[241] We discussed this idea of precipitously losing minute volume due to **AutoPEEP** and the high-pressure limit back already and will do so again when we get to **Alarms**

[242] Ragaller & Richter, 2010 - Not only do they provide an overview of the acute lung injury/ ARDS disease process, they also discuss this whole vent strategy and summarize research to date (at least as of 2010)

There are quantitative criteria for acute lung injury and/ or ARDS (depending on how we choose to define it), but that isn't necessary for our field treatment. Given our capabilities in the transport setting, we generally identify a patient who needs this vent strategy from a report per sending facility or suspicion based on clinical progression of the illness. There are also many recommendations to use this strategy for all patients who don't fit any other category.[243] In this sense we could think of the lung injury approach as we outline it here as a general strategy. We'll come back to that idea at the end of this section, but for now let's move on to the details. As we noted at the start, this lung injury strategy includes low **Tidal Volume**, higher than normal PEEP, and a focus on recruitment. Let's discuss each of these in turn and give some specific guidance.

Starting tidal volume for these patients should be 6ml/kg **Ideal Body Weight**, but we may get as low at 4ml/kg eventually. This recommendation is from a classic ARDSNet study which compared a tidal volume of 6ml/kg to 12ml/kg and determined that lower tidal volume resulted in better outcomes for these patients.[244] While it may seem that 6ml/kg and 12ml/kg represent two extremes and it could be tempting to rationalize that 8 or 10ml/kg probably isn't all that bad, we do know that 6ml/kg is OK and the rest is still up to debate at this point.[245]

In addition to low tidal volume, we go up on PEEP to improve both **Oxygenation** and **Ventilation**. As previously discussed, PEEP facilitates oxygenation by increasing **Alveolar Surface Area**, facilitating gas exchange through the expiratory side of the breath cycle and eventually, with time, facilitating movement of fluid out of the alveoli.[246] As for ventilation, PEEP actually helps to reduce **Dead Space** allowing for less wasted ventilation and better clearance of carbon dioxide.[247] Just know that this effect is limited and that PEEP taken too far can contribute to more dead space. Also recognize that when titrating PEEP, smaller changes with more time in between are less damaging to the alveoli and we likely won't be able to fix these patients in the time we spend with them during transport (i.e., don't get overly aggressive with increasing pressure over a short amount of time). And last thing, consider titrating PEEP alongside FiO_2 in a stepwise fashion as outlined in the charts below:[248]

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[243] And in the case of two-strategy recommendations, it is either this lung injury approach or an **Obstruction** strategy that make up the choices

[244] The ARDS Network, 2000 - Much of the data we have on contemporary vent management comes from this group of researchers and subsequent investigations by other folks based on their research

[245] Sahetya & friends, 2017; Burrell, 2018 - And for a more detailed discussion of this, take a look at this article (summarizes different arguments for why tidal volume ought to be tightly controlled in ARDS) and that review of another paper (which sought to investigate this idea of using a tidal volume in that middle area between 6ml/kg and 12ml/kg)

[246] This was discussed in **Oxygenation**

[247] Robertson, 2015; Murias & friends, 2014 - PEEP can help reduce dead space, but only up to a certain point; after that PEEP actually contributes more to the problem

[248] NHLBI ARDS Network, 2005 (image); NHLBI ARDS Network, 2004 - The study cited shows that either of those two approaches is appropriate; in fact, they modified the study in process to test even higher PEEP – we've left that out just to keep things a little simpler, but also because there is ongoing debate as to whether or not PEEP is the best strategy by which to improve outcomes in these patients; and then more on this in **Optimization of PEEP**

OXYGENATION GOAL: PaO_2 55-80 mmHg or SpO_2 88-95%
Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations such as shown below (not required) to achieve goal.

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

Another really important component of our lung injury strategy is alveolar recruitment. This is a concept that we've talked about some, but we'll get into it more here.[249] Recruitment is the idea that we can actively re-inflate collapsed or underinflated alveoli as we drew out in our previous discussion of PEEP.[250] One component of the acute lung injury/ARDS disease process is that the alveoli are particularly susceptible to both barotrauma/ volutrauma and stress due to repetitive expansion and collapse. By slowly filling the alveoli with air and then using small volumes of air with each breath, we maximize usable space within the lungs and avoid causing damage. PEEP allows us to make this happen (i.e., it maintains recruitment through this process) and in turn we may have to sacrifice net movement of air per a permissive hypercapnic approach (i.e., we sacrifice some degree of ventilation to maximize oxygenation).

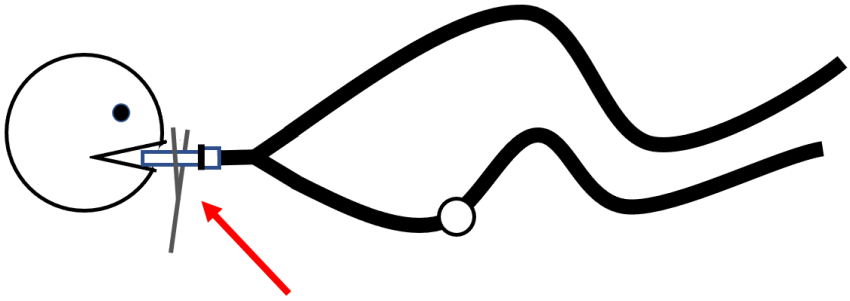
A few things about all of this: incomplete oxygenation in the ARDS patient is often due to ventilation/ perfusion (V/Q) mismatch and under-perfused regions of lung.[251] We previously said that the parameters to improve oxygenation are **Fraction of Inspired Oxygen**, **Positive End-Expiratory Pressure**, and **Inspiratory Time**. Even if we have all of these things optimized, we may still be short of our oxygenation goals and that's just part of the disease process. Further fixes for that ventilation/ perfusion (V/Q) mismatch would include time (i.e., having patience), minimizing dead space, and maintaining adequate ventilation.[252] While we often hear ARDS strategies described with a component of permissive hypercapnia, that idea is questioned by some and may go contrary to our overall goals.[253] There are varying opinions on this, however, so initiate a dialogue with sending or receiving facilities if specific questions about management of these patients becomes an issue.

Getting back on track with the idea of recruitment, if alveoli are stented open with PEEP and then the vent circuit is disconnected, those alveoli go back to a baseline, deflated state. In a normal lung there are forces that maintain recruitment to prevent this loss and we can also re-recruit alveoli on the order of seconds to minutes. This means that for the normal patient it isn't a huge deal to be worried about losing recruitment. Just get them on the vent again, add a bit of PEEP, and the situation is back where it was with no real negative outcome. With the lung injury patient, however, it can take hours to recruit alveoli. If recruitment is lost with

[249] And then we will talk about recruitment again when we get to **Recruitment Maneuvers**
[250] This was way back in **Positive End-Expiratory Pressure**
[251] Radermacher & friends, 2017 - This paper goes into lots of detail on how the gas exchange process is affected in ARDS and how treatments have changed over time as new evidence emerges
[252] To review these ideas: we talked about both the time factor and how alveolar dead space can be caused by ventilation/ perfusion (V/Q) mismatch in **Hypoxic Pulmonary Vasoconstriction**; we discussed **Dead Space** previously in that section and then again just a moment ago when we mentioned that PEEP can help to reduce dead space
[253] Modotto & friends, 2020 - This paper found neither benefit nor harm with hypercapnia in ARDS

these patients, all of that progress towards better oxygenation and ventilation can be lost, and the patient may deteriorate very quickly.

With that in mind, it is important to keep the system that extends from the vent to the patient's alveoli intact at all times. When we do have to break the system, such as when we transfer the patient from our machine to the hospital's machine or vice versa, we can maintain recruitment by clamping off the endotracheal tube.[254] The point is to prevent pressure at the alveoli from dropping below PEEP. In an ideal world, we'd use an ECMO clamp (which ensures a good clamp/ seal) at end-exhalation (to prevent high pressures if a breath is given immediately on reconnection).[255] And here is what the technique looks like:



clamp endotracheal tube with hemostats before disconnecting (consider using a 4x4 to pad things so that the teeth on the hemostats don't damage the tube)

Last thing to mention with this lung injury strategy is minute volume. We mentioned already that we start at a tidal volume of 6ml/kg and may need to go down to 4ml/kg. With higher PEEP we increase overall airway pressures and therefore that 6ml/kg tidal volume on top of a higher PEEP (up to 20cmH_2O in some cases) means we might run into a high **Plateau Pressure**. If we notice plateau pressure encroaching on our safe limit of 30cmH_2O, then we can dial the tidal volume down to 5ml/kg and then to 4ml/kg. Dropping our tidal volume to 4ml/kg will reduce minute volume and increase $EtCO_2$, but let's quantify that difference in minute volume with an assumed patient of 65kg ideal body weight:

MV goal = 6.5L

TV = 4ml/kg x 65kg
TV = 260ml

MV calculated = TV x RR
MV calculated = 260ml x 17/min
MV calculated = 4420ml
MV calculated = 4.4L

[254] Sidenote on this clamping technique: this is often discussed in transport medicine and has theoretical benefit given our understanding of the pathology, but there is no concrete evidence that doing so has an impact on outcomes

[255] Bulleri & friends, 2023 - These guys experimented with different clamps and at different times in the ventilatory cycle to identify the preferred technique; alternatively we could clamp whenever in the cycle or even at end-inhalation when in **Pressure Control** or if we place a patient on it before returning them to the vent (again, to avoid accidental high pressures), but that gets to be a bit confusing; also note this is a recommended practice change from Version 1 of this book and courtesy of insight during an in-depth discussion with Melody Bishop, RT

And to maintain our MV goal, let's see what kind of RR we would need:

MV goal = TV x RR
6.5L = 260ml x RR
6.5L / 260ml = RR
25 = RR

So to maintain our minute volume goal with a tidal volume of 4ml/kg we need a respiratory rate of 25 for the adult patient. With pediatrics (when a rate of 25 is too slow), we just go up on respiratory rate as much as we can to meet (or exceed if in volume control) our minute volume goal. Consider doubling respiratory rate, or using the high end of normal for a given age range, or just titrate up from a normal rate. The limiting factor will be comfort and exhalation (i.e., monitor for AutoPEEP to ensure full exhalation).[256] And as we mentioned just a moment ago, there is some evidence that permissive hypercapnia (i.e., a high $EtCO_2$ related to a lower minute volume) is alright for these lung injury patients, but the data isn't super clear at this point.[257]

To put it all together: lung injury represents a spectrum of disease that primarily impacts the integrity of the alveolar and capillary walls; this results in increased permeability, movement of large molecules and fluid into the alveolar space, and further damage from an inflammatory response. Vent strategy is focused on low tidal volume starting at 6ml/kg (down to 4ml/kg if needed) to avoid barotrauma, high PEEP to both maintain recruitment of alveoli and displace fluid, maintenance of recruitment at all transfers in order to avoid rapid deterioration, and an increase in rate to maintain minute volume (possibly with a concurrent strategy of permissive hypercapnia).

And one last thing to mention about this strategy. We said just a moment ago that lots of folks recommend a two-strategy approach to ventilation in which we use either this lung injury approach or an obstruction approach. We have a general vent strategy for routine ventilation and then specific strategies for certain patient types. The differences between our general strategy (which is similar to a general lung-protective one) and this lung injury strategy is related to recruitment of alveoli (both using high PEEP and being super careful to not lose it) and the idea that we may need to go down on tidal volume to 4ml/kg. Both of these things are totally OK in the normal patient that we ventilate using the general strategy, it's primarily a matter of emphasis. If it makes things easier to default to this lung injury strategy in all cases that don't warrant one of the others, that's completely acceptable (just may not be optimal…).

Other Potential Strategies

This overview of vent strategies addresses four markedly different situations that we often come across in the transport setting, but there are other injuries or disease processes that might also warrant specific adjustments to these normal options. While we could theoretically compile a list of all the possible things and work out an algorithm to address each one in turn, that gets a little cumbersome and would result in a hefty protocol that might be difficult to navigate through when time is of the essence. The idea is to work towards an understanding of how the body responds and how the vent does its thing so that we can make changes and anticipate the results that will come of any adjustment away from normal. But just to mention a few examples without going into the same level of detail as we did just now, consider the following situations.

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[256] A few notes on this: we talked about this overshooting of minute volume with pediatric patients in the section on **Volume Control** and will do so again in both **More on Pediatric Ventilation** and the **Appendix**, also, the likelihood of transporting a pediatric patient with ARDS is slim and low-frequency, so ask for help and/ or consult with a specialist on this

[257] Just to clarify: the idea here is that permissive hypercapnia is tolerable for the lung injury patients, not that it provides an extra benefit

In the patient with a head injury or traumatic brain injury, we often choose to aim for an $EtCO_2$ low-of-normal to what we'd typically use for a standard patient.[258] While we don't necessarily hyperventilate these patients anymore, we could adjust **Minute Volume** to a tighter $EtCO_2$ goal of 35-40mmHg by going up on either **Tidal Volume** (preferred) or **Respiratory Rate**. Carrying on with this idea: we want to simultaneously avoid hypoxic events and minimize the effects of too much oxygen.[259] We also want to find a balance between maintaining comfort to avoid increases in intracranial pressure and being able to perform neuro assessments to track patient progress. All that said, consider using tighter parameters for **Comfort** and **Oxygenation** and/ or reassessing these patients more often.[260] Last thing on the head injury idea: there was once upon a time an idea that **Positive End-Expiratory Pressure** should be limited (and even, in some cases, dialed down to zero or ZEEP) - that idea hasn't held up and it is generally considered just fine to use normal levels of PEEP with TBI and/ or head injury.[261]

In the pregnant patient we might utilize a **Fraction of Inspired Oxygen** of 100% to maximize oxygen delivery to the fetus.[262] Since many services don't have the capability of fetal monitoring during transport, this is a way to ensure that we don't have a hypoxic injury or put any undue stress on the fetus. We also need to consider an increased minute volume goal for the patient (which may mean an $EtCO_2$ goal low of normal, somewhere in the 30-35mmHg range), as we have a baby to consider as well.[263] Another consideration is patient positioning. In the ventilated pregnant patient we not only have decreased preload due to positive-pressure ventilation, we could see that drop in cardiac output compounded by pressure of the fetus on the inferior vena cava. So either turn the patient to a lateral recumbent position or displace the gravid uterus over to the side.

Significant chest trauma is another one. We'd like to treat these patients via the **Lung Injury** strategy, but we may also be concerned with hemodynamics and want to use the **Hypotension** strategy. Those two are at odds with one another (low tidal volume and high respiratory rate for lung injury, high tidal volume and low respiratory rate for hypotension). Maybe we forgo the hypotensive strategy and choose the lung injury one, but get aggressive early on with vasopressors and fluids and/ or blood products in anticipation that a hypotensive state may be precipitated by our strategy. Or maybe we go with a strategy more in line with the hypotensive strategy, but start with lower PEEP and leave FiO_2 at 100%. There is no right or wrong here and it depends a lot on how the patient presents in that particular situation and what resources we have to work with.

On a tangent to this chest trauma idea: if a patient develops a tension pneumothorax en route, the best thing we can do may be to take the patient off the vent.[264] Not take them off the vent and bag them, but take them off the vent and don't breathe at all for them until we fix that problem. Positive-pressure ventilation can tension a pneumothorax very quickly and we want to avoid making things worse.[265] While this is more of an issue with pneumothorax caused by acute trauma and it isn't unheard of to see patients in a relatively stable condition while ventilated with a pneumothorax in the hospital, in transport we will likely notice a pneumothorax after it has started to tension and that is a true emergency. So disconnect the vent, decompress (or place a chest tube/ perform a finger thoracostomy), and then get the patient back on the vent. Because of this, we may consider keeping all patients with the potential for pneumothorax on a FiO_2 of 100%. That allows us more time to perform the procedure, if a pneumothorax develops, before the patient desaturates.

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[258] Godoy & friends, 2017 - Detailed overview of ventilatory management in traumatic brain injury with a review of research that has been done to date

[259] Discussed in a bit more detail in our YouTube video *Oxygen Haterz*

[260] Refer back to **Oxygenation** for a discussion of the negative effects of oxygen and then to **Comfort** and **Asynchrony & Dyssynchrony** for strategies to work towards ventilator synchrony

[261] Iavarone, 2025 - As with all things, the details on this are multifactorial and complicated

[262] While this practice is fairly routine, the data to support it not so clear; see our video *Oxygen Haterz* for more

[263] Wingfield, 2012a; LoMauro & Aliverti, 2015 - The idea of a lower $EtCO_2$ goal with the pregnant patient is suggested in the video; the physiology behind it is discussed in the article

[264] Flowers & friends, 2019 - Per Boyle's Law (increase in volume with a decrease in pressure), we can cause this tension by taking a stable pneumothorax to elevation

[265] Wingfield, 2012b; Hsu & Sun, 2014 - Both of these sources discuss pneumothorax in the mechanically ventilated patient; the first discusses this idea of removing these patients from the vent immediately, the second is an overview of the idea and outlines a simple algorithm for managing these patients

A patient with cardiogenic pulmonary edema from left-sided heart failure may warrant more PEEP while the patient in right-sided failure may benefit from lower levels.[266] And while it may make sense that a high FiO_2 could mitigate the effects of **Hypoxic Pulmonary Vasoconstriction** in either of these groups, there is some risk to that strategy.[267] Folks with chronic obstructive pulmonary disease may ought to have oxygenation tightly controlled due to the potential effects of oxygen.[268] We could even argue the case for a specific toxic-exposure strategy: some combo of lung injury plus or minus **Acidosis**, depending on the agent and route of exposure.

Following along with this idea, there are also times that we have a patient who presents in a way that results in competing strategies. Say, for example, a patient who is both acidotic and hypotensive. We know that the **Acidosis** strategy warrants an aggressive increase in **Minute Volume** to avoid acute decompensation, but also recognize that this is contrary to the **Hypotension** approach and will increase **Mean Airway Pressure** which potentially results in worsening hypotension. At this point we need to recognize that the vent is not the only tool we have to fix the patient - maintain ventilation at an increased level to avoid worsening of the acidotic state and utilize other interventions to address hypotension.

And then we'll talk about pediatric strategies more specifically in **More on Pediatric Ventilation** later, but there could also be the case for different strategies in certain congenital heart defects.[269] For example, in a left-to-right shunt (i.e., some sort of septal defect) we might limit oxygen delivery, allow permissive hypercapnia and utilize more PEEP; but in right-to-left shunt (i.e., Tetralogy of Fallot or tricuspid atresia) we might be more aggressive with oxygen, ventilate more to a lower $EtCO_2$, and withhold PEEP. This sort of thing is a bit beyond the scope of this manual, but we mention it simply to demonstrate that vent strategies can get quite niche for certain populations.

It quickly becomes evident that there are a number of cases that don't quite fit the mold by which we try to simplify vent strategies. And that's totally OK. The templates are there as frameworks from which we then consider the specifics of each patient, one at a time. The important thing is to know what impact any vent change will have on the patient depending on how (s)he presents in a given situation. There are lots of cases in which there isn't a straightforward answer, but as long we don't make things worse by titrating things the wrong way, all is good.

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[266] This distinction is touched on again in **Therapeutics for Hypotension**

[267] Kuhn & friends, 2016 - This article reviews mechanical ventilation strategies for patients with heart failure; while it focuses on the extreme end of these patients (i.e., decompensated heart failure), some of the ideas could apply to congestive heart failure patients on a general sense

[268] Swaminathan, 2015 - Short discussion of oxygen administration in COPD with a focus on investigating some of the common misconceptions; also see the *Oxygen Haterz* video we made

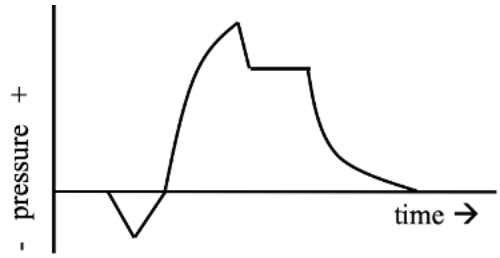
[269] This is something we have looked into and built some content on at the request of an agency, but have not yet verified or had reviewed by anyone who works in the NICU space; feel free to take a look anyways: *Mechanical Ventilation with Cardiac Defects*

Additional Concepts Round One

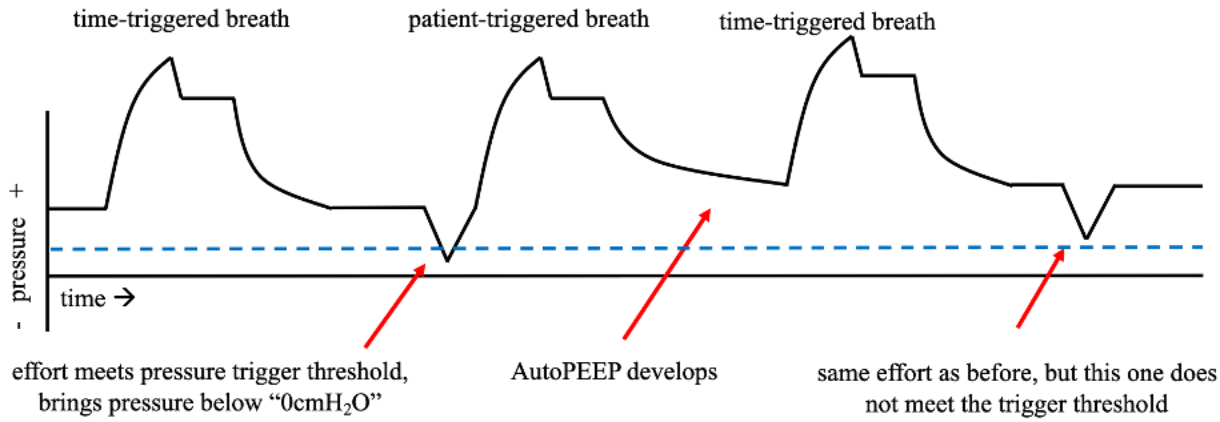
Before we get to putting these various strategies into action, there are just a few more concepts to cover. We've touched on some of these in passing already, but let's take the time to look at them in a bit more detail so we are better prepared when it comes time to lay everything out in a plan of action.

Triggers

Triggers are the thresholds by which the machine knows when a patient is trying to breathe on his or her own. We first tried to communicate this idea via the following graphic:



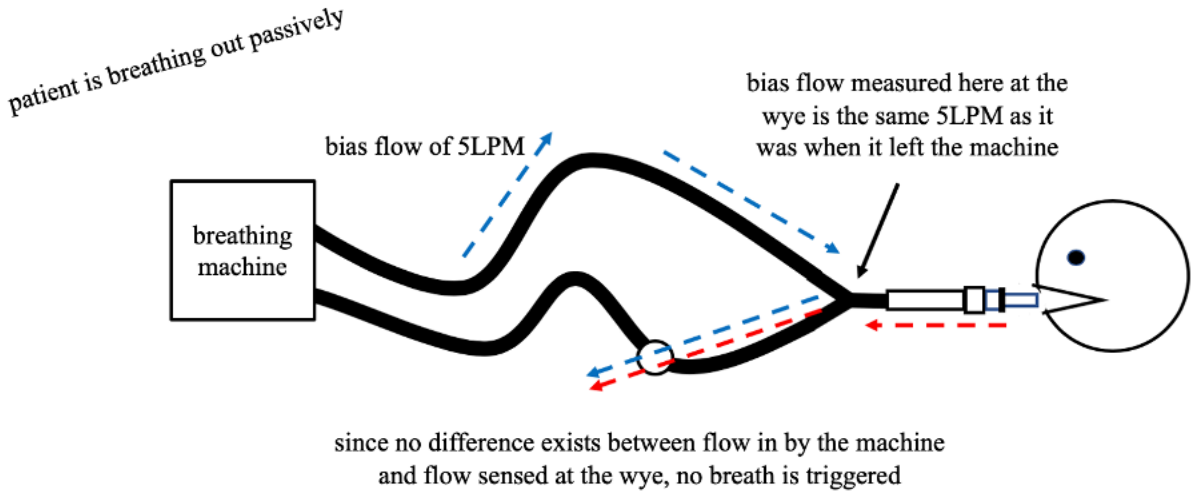
And then we footnoted the idea that that downward dip in pressure at the start of the waveform is more a sketch of convenience than an accurate representation of how things normally occur.[270] In most cases the trigger that makes the machine recognize patient effort is based on flow rather than pressure. While some machines will allow us to use pressure triggers (normally around -1cmH_2O), this isn't commonly used. Pressure triggers have been shown to be more difficult for patients to overcome (at least with older model ventilators). In addition, the pressure trigger is relative to what we have dialed in for **Positive End-Expiratory Pressure**. This means that in the event of **AutoPEEP** there is an extra threshold that must be overcome:

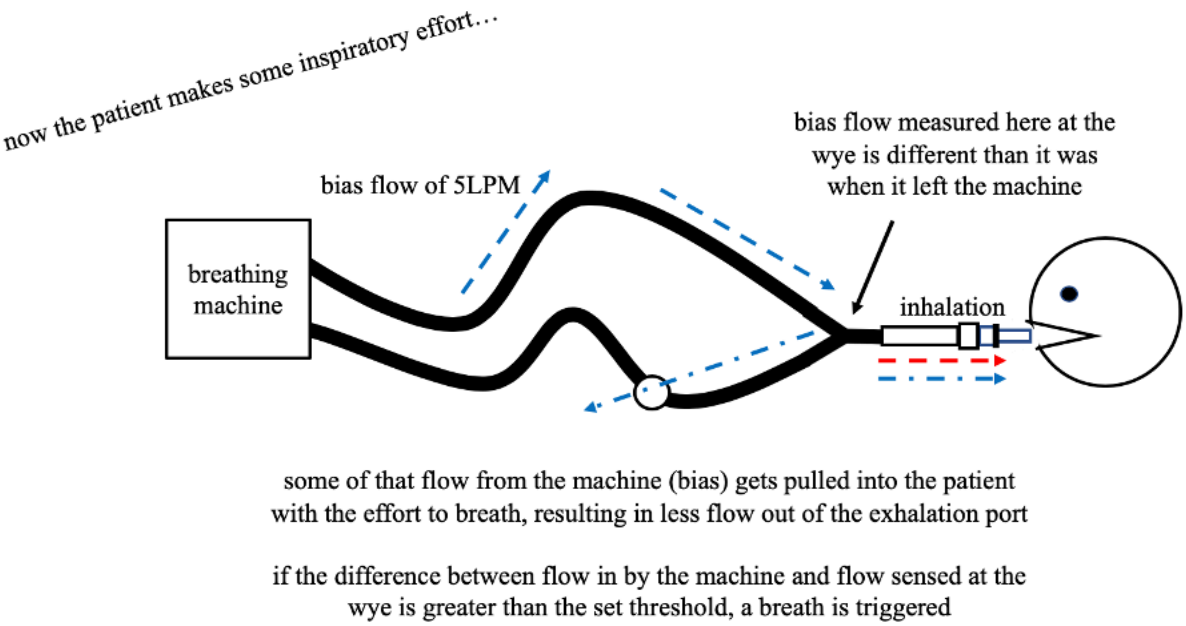


So pressure triggers are a thing as we initially drew it out, but not the most common thing. We sometimes do use pressure triggers in cases of auto-triggering (i.e., when we see too many triggered breaths due to things other than patient effort, such as bumpy roads in an ambulance or turbulence in an aircraft), but for the most part we stick with flow triggers.[271] To measure flow changes against a zero reference (the pause between breaths is a zero-flow state) the machine uses a concept called bias flow. Bias flow is a baseline flow of air into

[270] We first drew this waveform out when we were talking about **Modes of Ventilation**, specifically the section on **Assist Control**
[271] Hess, 2005 - While we could utilize pressure triggers to mitigate worsening AutoPEEP with increased patient respiratory effort (assuming an initial flow trigger), we prefer to address the cause of discomfort or meet the patient's demands rather than ignoring it altogether; more on that in **Asynchrony & Dyssynchrony**

the system against which changes are measured. So when the machine says there is no flow going into the system, there is actually some flow going in, but it gets factored out. Let's draw it out with an assumed bias flow of 5LPM just to see how it works:[272]





The machine does this bias flow thing because it makes it easier to measure patient effort. Bias flow can either be set manually or adjusted automatically by the machine based on what we have set as the flow trigger.[273] It's not something we routinely consider in transport, but if we are working with a machine in which bias flow is manually controlled it's worth knowing that we can't have a trigger set higher than our bias flow.

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[272] Yartsev, 2023j - For an alternative explanation of how this flow trigger works (plus much more detail on triggers in general), take a look at this article; also go back to **Ventilator Anatomy**, if needed, for a review of this sketch and all the associated parts of the setup

[273] Chatburn & Mireles-Cabodevila, 2013 - For a brief discussion of bias flow, scroll down to *The Operator Interface* and then *Flow* within this chapter

So if our machine has a bias flow of 5LPM and we want a trigger higher than that, we would have to increase the bias flow.

A normal flow trigger in the hospital setting is 1-2LPM. This value is also referred to as sensitivity (i.e., a sensitivity of 2LPM would indicate a flow trigger of 2LPM). For reference, the normal mean flow produced by a human at rest is about 15LPM with a peak of about twice that.[274] Which means we could theoretically dial our flow trigger much higher than the 1-2LPM that we normally use and still expect a patient to trigger breaths. As we discussed previously, we titrate sensitivity to allow patient effort to trigger breaths, but also to prevent other miscellaneous input from accidentally causing triggers.

To expand on this idea, let's consider things sequentially. If breaths are being triggered, first thing is to try and identify what input is causing the triggers. If it seems likely that the input is patient effort, all is well. If, however, there is something else that is causing the trigger (i.e., auto-triggering or false trigger), then we troubleshoot that.[275] Bumpy roads or turbulence (especially with the vent circuit laying on the floor of the vehicle) and accidentally kicking or stepping on the circuit are common causes of auto-triggering.[276] The preferred approach is to fix those things directly and solve the problem without changing the sensitivity on the machine. If, however, we cannot alleviate the accidental triggering of breaths, then we can increase the trigger sensitivity or consider a different type of trigger (i.e., change from a flow trigger to a pressure trigger). The idea is to simultaneously prevent breaths from being accidentally triggered and keep the trigger threshold as low as possible so that it is easier for the patient to meet the trigger.[277]

To summarize triggering: triggers are thresholds we set to allow the machine to know that the patient wants to take a breath. We most commonly use flow triggers, but some machines allow for pressure triggers as well. Flow triggers are based on and limited by bias flow. A sensitivity of 1-2LPM is commonly used in a hospital setting. Auto-triggering happens when the trigger is inadvertently met by something other than patient effort to breathe. Fixes to auto-triggering include mitigating the cause of the inadvertent trigger, increasing the sensitivity, or trialing a different type of trigger.

Overbreathing

Just to close the loop on one idea that we mentioned back in the section on **Vent Parameters, Round One**, let's consider what to do if we think the patient is breathing too fast and/ or in a way that goes contrary to how we want to manage **Ventilation**. Say we have a patient breathing faster than we'd like and, as a result, our $EtCO_2$ is low. Normally we'd decrease **Respiratory Rate** to drop **Minute Volume** and get that $EtCO_2$ back in range, but now we have to consider these patient-triggered breaths and the fact that decreasing the parameter on the vent won't decrease the overall rate.[278] First thing to think about is that an increased rate of breathing is the body's normal response to lots of things: decreased pH or increased $PaCO_2$ (as we discussed in **Acidosis**), pain, fluid in the lungs, irritants in the airways, anemia, cardiac ischemia, etc.[279] Next we need to consider if it's a problem or will have negative consequences for our patient - in most cases an increase in minute volume is well-tolerated. So before we label this overbreathing as an anomaly and decide that something needs to be done, consider that it may actually be an appropriate response and/ or a tolerable consequence.

Related to this idea, if the concern is minute volume in a general sense and not necessarily respiratory rate, we can simply decrease **Tidal Volume** or consider switching from **Assist Control** to **Synchronized Intermittent Mandatory Ventilation** to try and bring minute volume down. It's important to remember that

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[274] Yartsev, 2023j - We cited this website on the last page, but he specifically outlines the norms for both in-hospital flow triggers and flow produced by normal respiration which we cite here

[275] And for how to do that, exactly, look to **Asynchrony & Dyssynchrony**

[276] This tends to happen less often with newer vents than with older ones, as noted at the end of **Modes of Ventilation** in **And Beyond…** when we talked about the arguments in favor of **Synchronized Intermittent Mandatory Ventilation**

[277] This concept of maintaining patient effort was first discussed back in **How is Positive-Pressure Different?**

[278] As discussed in **Respiratory Rate** the difference in notation here is that measured or observed respiratory rate (also described as frequency) is the total number of breaths, both time-triggered and patient-triggered, the rate we set on the machine only describes the time-triggered breaths

[279] Murphy, 2017a; Alexander, 2016 - The first is one video of three part series on control of breathing, feel free to check out the others for more; the second is an article about tachypnea in general, provides some insight on overlooked causes of tachypnea

minute volume is the product of both respiratory rate and tidal volume. Even though we routinely modify respiratory rate to address an excessive minute volume, decreasing tidal volume is also an appropriate strategy. But if we don't have an identifiable cause and rate is the thing we want to fix, the next considerations to address too many patient-triggered breaths are **Comfort** and **Triggers**. We talked already about both of these ideas, so we won't get into too much detail here.[280] The general idea is that if patient-triggered breaths are the issue (i.e., too many of them happening), we can fix that by either reducing discomfort, manipulating the trigger threshold itself, and/ or avoiding accidental triggers.

There is one more thing to note about this. We mentioned back in **Basic Modes of Ventilation** that both controlled mechanical ventilation and intermittent mandatory ventilation are modes that do not have a mechanism for patient-triggered breaths. We said then that some ventilators allow us to approximate these modes (i.e., ignore patient effort to breathe) and theoretically that would be an option in the case of overbreathing. That said, this strategy is likely to be very uncomfortable for the patient and would probably require heavy sedation and maybe even paralysis to work. We generally don't want to simply ignore patient efforts, rather we would prefer to do things to promote synchrony and work alongside the patient.

Back to our discussion; if we've ruled out all of these situations and the patient is still breathing too fast and we've already considered both comfort and triggers, what else is there to do? First thing is to verify that the increased respiratory rate (the cumulative total of our set rate plus patient-triggered breaths) is, in fact, a problem. It may be the case that the patient is breathing fast, but with no identifiable negative outcome. Reassess the patient with a focus on the **Three Big Things** and then decide if it's still a problem that needs to be addressed. If so, we have a few options: **Positive End-Expiratory Pressure**, increasing tidal volume, and paralytics.

Adding PEEP can be more comfortable for a patient (which may lead to fewer triggered breaths) and may also reduce minute volume while keeping the lungs open and participatory in ventilation. Just recognize that in **Volume Control** ventilation we will need to reevaluate pressures to ensure that the alveoli remain safe (i.e., that our **Plateau Pressure** is still acceptable). Moving forward, there is also the idea that increasing tidal volume can lead to less dyspnea or air hunger due to an effect on chemoreceptors in the lungs.[281] And then if we increase tidal volume and exceed the minute volume needs of the patient, they will respond with a slower rate of breathing. While this is a bit counterintuitive, it could be worth trialing. And at the extreme end of things we could administer paralytics, but we prefer to reserve that strategy for life-threatening situations.

As a related example, let's say we have a ventilated patient who suddenly experiences acute bronchospasm or has an anaphylactic reaction. Per our **Obstruction** strategy we would like to decrease our I:E ratio by decreasing both **Inspiratory Time** and **Respiratory Rate**. So we do that on the vent, but the patient is still triggering breaths for an actual or calculated I:E ratio higher than we'd like and with the result that **AutoPEEP** starts to develop.[282] At this point the rate dial on our machine is not the primary way we control the overall rate and we need to think about other ways to bring the patient's intrinsic rate down to achieve our goals. Similar to what we said above, strategies here would include further decreasing inspiratory time, addressing comfort, making sure triggers are appropriate, and adding PEEP.

So while we've discussed adjusting respiratory rate up to this point as if it were a parameter we have complete control over via the machine interface, recognize that the number of breaths per minute can exceed the value we have set. Patient-triggered breaths are generally of benefit to the patient, but if they threaten overall wellbeing or safety then we ought to take action to get things back on track.[283] Steps to fix this problem include addressing comfort, evaluating triggers, adding PEEP, trialing an increased tidal volume, and, as a last resort, paralysis.

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[280] But just to mention it: look to **Asynchrony & Dyssynchrony** in **Vent Graphics** to learn about those patterns, just keep in mind that not all dyssynchrony results in overbreathing and not all overbreathing means there is a concurrent dyssynchrony

[281] Banzett & friends, 2013 - For a very detailed discussed of this idea, take a look at this chapter in Tobin's textbook on vent management

[282] This is not to say that overbreathing always results in AutoPEEP, just that it can

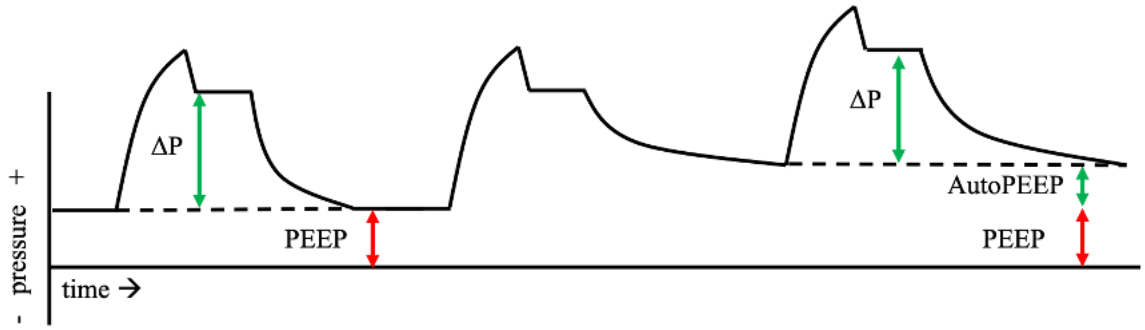
[283] This idea that patient-triggered breaths can be of benefit was discussed back in the section on **Comfort**

Driving Pressure[284]

Driving pressure is a term to describe how much we inflate and deflate the alveoli with every inhale and exhale on the ventilator. The idea is that too much opening and closing (inflation and deflation, up and down – however we want to term it) can put stress on the alveolar walls and cause damage.[285] This damage, in turn, leads to decreased diffusion of gases across the alveolar membrane. Driving pressure is the difference between **Plateau Pressure** and end-expiratory pressure (normally **Positive End-Expiratory Pressure**, but sometimes affected by **AutoPEEP**) and is sometimes referred to as delta pressure:[286]

$$\Delta P = Pplat - PEEP$$

As an image, it would look like this:



With our **Lung Injury** patients, we try to limit driving pressure as much as we can to a max of 15cmH_2O.[287] This is generally pretty reasonable, given that we use high PEEP and low **Tidal Volume** in these patients anyways. And if driving pressure is close to or above that upper limit, we can do **Recruitment Maneuvers** to try and utilize more lung, increase compliance, and drop driving pressure.[288] This approach may sound familiar and is often referred to as open-lung ventilation.[289] The basic idea is that we keep the lungs as filled as possible (i.e., alveoli inflated) throughout as much of the respiratory cycle as we can. This concept of limiting driving pressure and an open-lung strategy is specific to the ARDS population.

With that said, there may be the case for a comparable strategy in other patient groups, but the data is mixed.[290] The one downside of this limited driving pressure or open-lung approach is that it can be tough to blow off carbon dioxide as much as we'd want.[291] We said way back when that permissive hypercapnia is often a thing with ARDS, but that may not be the case with other patient groups. Another consideration here is PEEP – it is not a benign thing and we for sure need to consider the negative consequences of this approach before applying it to all patients. For now we have pretty good evidence that limiting driving pressure and utilizing high PEEP is a good thing with ARDS, but such a strategy may not be best for everyone.

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[284] Bugedo & friends, 2017 - Succinct overview of the concept of driving pressure and research done to date (as of a few years ago, at least)
[285] Grune & friends, 2019 - While it is commonly accepted that this inflation/ deflation cycle does cause damage (and we will assume it to be valid in our discussion), know that there is ongoing research on all of this (as shown in this article)
[286] Lewis, 2019 - That said, these two concepts (driving pressure and delta pressure) are distinct ideas, this video explains the difference
[287] Weingart, 2016a; Bauer, 2016b - Both podcasts look at a 2015 study on the subject of driving pressure
[288] We will discuss **Compliance & Resistance** and **Recruitment Maneuvers** in the next two sections
[289] Nickson, 2019b - We will get much more into this idea of open-lung ventilation shortly, but this page provides a nice overview of the concept to hold us over until then
[290] Sahetya & friends, 2019; Schmidt & friends, 2018 - The first argues in favor of limiting driving pressure in non-ARDS patients; the second posits that there is not mortality benefit
[291] To say it another way: by limiting driving pressure, **Ventilation** may be affected and **Minute Volume** may be less than we might want

Compliance & Resistance[292]

Compliance is a measure of how much the lungs fill per unit of pressure put into the system. In math terms it looks like this:[293]

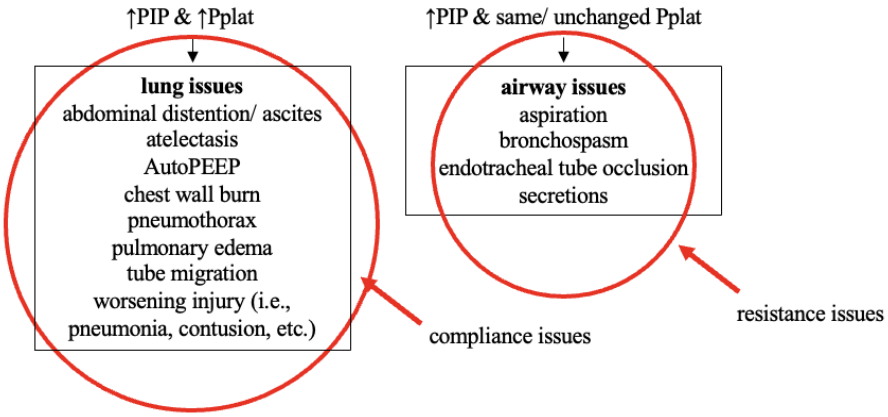
$$\text{compliance} = \frac{\Delta V}{\Delta P} = \frac{TV\ or\ Vte}{(Pplat - PEEP)}$$

While normal compliance (healthy and breathing spontaneously) is somewhere in the neighborhood of 100ml/cmH_2O, we often see values much smaller than that in our ventilated patients. The best way to utilize compliance during transport is to keep track of trends: increasing compliance is good, decreasing compliance is bad. If we do something that results in poorer compliance, maybe second guess whatever that change was; if we do something that results in better compliance, high fives are warranted. Acute causes of decreased compliance would be a worsening pneumothorax, inhibition of chest wall expansion, chest wall rigidity caused by certain medications, increasing **Tidal Volume** beyond the capacity of the lungs at that given time, etc.[294]

A related term is resistance. Resistance and compliance are often discussed together under the umbrella terms of respiratory mechanics or pulmonary mechanics, that's why we talk about it here.[295] Now the algebraic expression of resistance isn't quite as straightforward as for compliance and we often simplify it by assuming that flow equals 60LPM, so we're just going to skip on ahead and note it like this:

$$\text{resistance} = PIP - Pplat$$

Resistance, in this simplified manner, is the limitation to air movement that must be overcome to arrive at a state in which air in from the machine gets to the alveoli. Assuming plateau pressure remains constant, resistance is represented by peak inspiratory pressure. This means that we can approximate changes to peak inspiratory pressure to signify changes to resistance. So things like kinks in the tubing, biting on the tube, excessive secretions, etc. that are causes of increased peak pressure and unchanged plateau pressure correlate with an increase in resistance:[296]

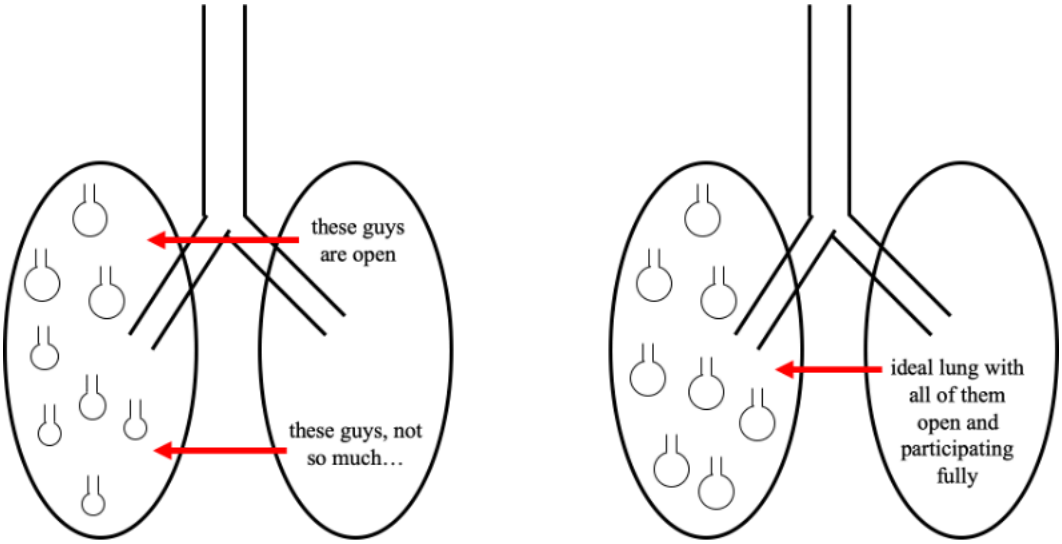


[292] Trainor & friends, 2019 - This video reviews both compliance and resistance in a succinct and straightforward way
[293] To be more specific, this is what we would call static compliance; we won't get into dynamic compliance here
[294] All of these high **Peak Inspiratory Pressure**, high **Plateau Pressure** situations will be discussed in the section on **Watching Pressures**
[295] Hess, 2014 - And for much more on this concept of respiratory mechanics and all the other things included, refer to this guide
[296] Cassone & friends, 2019 - We will expand on this in **Watching Pressures**, but know for now that this graphic is a piece of an algorithm that we lay out when we get there – it just made sense to include it here to differentiate these two concepts

And we mentioned already that the alternative strategy in **Pressure Control** ventilation when we don't have peak inspiratory pressure or plateau pressure to guide us is to look at exhaled tidal volume and exhaled minute volume to gauge when these things are happening (a drop in volume will indicate an increase in resistance or decrease in compliance). We can also look at a quantitative value for compliance (if available to us on a given machine) or see how flow is changing from breath to breath.[297]

Recruitment Maneuvers

A recruitment maneuver is a technique that attempts to get more alveoli involved in the breathing process.[298] During ventilation, and even at health, there are portions of the lung that are open or participatory and others that are closed down or non-participatory (or maybe just less-than-optimally-participatory), and we can do things to gain access to those clamped-down alveoli to improve both **Oxygenation** and **Ventilation**:



In a general sense, lots of things could qualify as recruitment maneuvers: prolonged inspiratory holds, higher **Positive End-Expiratory Pressure**, high-frequency oscillation ventilation (HFOV),[299] airway pressure release ventilation (APRV),[300] **Prone Ventilation**, spontaneous breathing, etc. Basically anything that can help open those non-participatory alveoli falls into this category.[301] That said, we tend to consider recruitment maneuvers to be either the prolonged inspiratory hold or the stepwise approach, so we will stick with those two ideas moving forward.[302]

A recruitment maneuver can be used in any patient group, but has been most studied with ARDS patients under the umbrella term open-lung ventilation. We can think of open-lung ventilation as a supplement to the lung-protective approach we described in **Lung Injury**. The focus of lung protection is smaller **Tidal**

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[297] Again, this in pressure control only; in volume control flow will generally be the same with each breath delivered

[298] Ragaller & Richter, 2010 - We talked about the advantages of getting more alveoli involved in the breathing process both in **Alveolar Surface Area** and **Oxygenation**; the article is an overview of ARDS management with a section on the idea of recruitment; it also refers to the idea of open-lung ventilation, which we will discuss momentarily

[299] Prost, 2011 - This is the only mention we make of HFOV, as it isn't routinely available in transport; the referenced video is an overview of it

[300] Farkas, 2017 - While **Airway Pressure Release Ventilation** isn't common in transport at the moment, chances are we will start to see more of it in years to come; navigate here for a discussion of this mode in the context of an open-lung approach (discussed on the next page); also refer to many more references at the bottom of the page

[301] Naik & friends, 2015 - This is an article that also discusses recruitment, but particularly the idea that breaths of various sizes (whether intentional via vent management or spontaneous via patient effort) further contribute to recruitment

[302] Hartland & friends, 2015 - This paper both discusses recruitment maneuvers and describes their use in non-ARDS patients under anesthesia

Volume and keeping **Plateau Pressure** within normal limits. The open-lung idea adds to that a component of utilizing recruitment maneuvers to get more alveoli involved.[303] While these two ideas (lung-protective and open-lung) are not universally defined and there is some overlap, the general idea holds true.

Recruitment maneuvers have been shown to increase oxygenation and improve ventilation/ perfusion (V/Q) mismatch, but outcomes in terms of mortality and days on the vent seem to be unaffected or even worse.[304] To further complicate things, when we do try and get into the weeds as to how we should perform a recruitment maneuver, techniques vary significantly and there are potential adverse effects. So here's where we stand on this: more data is clearly needed, but there is low-quality evidence that some benefit exists from performing recruitment maneuvers in ARDS patients, particularly as part of an overall open-lung strategy.[305] Translating that to the non-ARDS patients who are simply hypoxic is a bit tough, as there isn't much data out there and we can often fix the issue by way of things we've already talked about (**Fraction of Inspired Oxygen**, **Positive End-Expiratory Pressure**, and **Inspiratory Time**) and ensuring adequate perfusion.[306]

But let's say we do want to do a recruitment maneuver anyways. Maybe we are struggling to oxygenate a patient, or we forgot to clamp the endotracheal tube on transfer of an ARDS patient to our vent, or we want to try for better compliance and/ or decreased **Driving Pressure**, etc.[307] First thing to know is that the maneuver can cause hemodynamic problems and we ought to be on the lookout for those to avoid decompensation. Just as we discussed back when we first got into **How is Positive-Pressure Ventilation Different?** and PEEP, an increase in intrathoracic pressure can drop preload and subsequently impact cardiac output. So monitor all the things and have hard limits in place for abandoning the maneuver.[308] Also, recognize the risk for causing a tension pneumothorax and consider that an underinflated tube cuff or uncuffed pediatric tube will render the maneuver less effective.[309]

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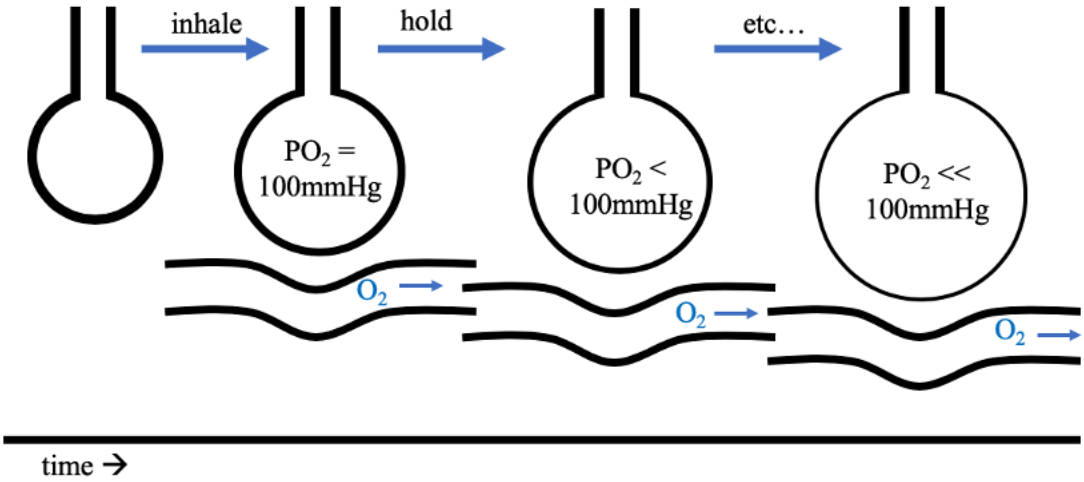
[303] Nickson, 2019b - Refer here for an outline-style overview of this open-lung strategy and its components
[304] van der Zee & Gommers, 2019 - This article describes much of the research that has gone into understanding recruitment maneuvers, particularly in the context of an open-lung approach
[305] Hodgson & friends, 2016 - Cochrane Review that summarizes the data on recruitment maneuvers in ARDS
[306] These steps were discussed in **Oxygenation**
[307] See **Compliance & Resistance** for a review of this concept
[308] Claire & friends, 2019 - And for suggestions on limits to use while performing one of these maneuvers and an explanation of the next technique (the stepwise recruitment maneuver), take a look at this short guide
[309] Chambers & friends, 2017 - This study primarily examined how exhaled tidal volume differed from delivered tidal volume with cuffed and uncuffed tubes, but it also looked at the effect recruitment maneuvers have on this difference

The simplest way to do a recruitment maneuver is via a prolonged inspiratory hold.[310] We posed a hypothetical situation at some point earlier on in this manual about why we don't just blow up the lungs and alveoli with oxygen and let it sit like that for a while.[311] We said then that we still have to consider the ventilation side of things, but the idea itself does have some merit. The value of a recruitment maneuver (again, this is as a prolonged inspiratory hold) is more in the ability to get alveoli open than in the inflow of oxygen for a sustained amount of time, as the amount of oxygen in that air quickly begins to drop as it diffuses into the bloodstream and we don't replenish the supply:



To perform a prolonged inspiratory hold as a recruitment maneuver, here's how it works: Put the patient in **Pressure Control**, set the pressure control parameter to achieve a goal pressure (keeping in mind that we may or may not need to factor PEEP into this), and perform an inspiratory hold for as long as we deem appropriate. Recommendations on specifics vary widely, but a pressure of 30-40cmH_2O for about 30s is often cited.[312] That said, there are many opinions that a hold beyond ten seconds doesn't provide any additional benefit.[313] There's also the question of how often we ought to or can perform these maneuvers. Most of the data out there is specific to in-patient settings with these maneuvers performed relatively infrequently, so this isn't something that would perform many times throughout transport, rather we may do it once after getting a patient set up on our vent.[314] We won't get into specifics beyond that, as this would be a better conversation to have with both the providers involved in a specific patient case and medical direction within a given agency.

Moving on to the next type of recruitment maneuver, we have the stepwise approach. We mentioned already that whenever we put more air into the lungs it seems advantageous to do so incrementally. Same goes for performing a recruitment maneuver. An alternative to the prolonged inspiratory hold would be a stepwise approach.[315] In **Volume Control** we would simply increase PEEP incrementally and over time; in pressure control we would establish a **Driving Pressure** that yields our goal **Tidal Volume**, then slowly titrate up on PEEP in a similar fashion.[316] There is a rendition of this approach called the Staircase Recruitment Maneuver

[310] Metz, 2016a - Video that shows a prolonged inspiratory hold in a set of lungs attached to a vent circuit
[311] This was in **Oxygenation**
[312] NHLBI ARDS Network, 2004; Hartland & friends, 2015 - While a few other references cited in this section also mention a prolonged inspiratory hold in line with these two articles, it seemed appropriate to note some things: this type of maneuver was tested by the ARDSNet folks and subsequently discontinued during the trial; the other study used a similar maneuver plus two others and found positive benefit with all of them prior to patients undergoing surgery
[313] Radermacher & friends, 2017 - Knowing that a prolonged inspiratory hold may not have benefit beyond ten seconds and given the fact that performing the maneuver in general can affect hemodynamics means we should probably limit the length of these types of recruitment maneuvers
[314] Hodgson & friends, 2016 - While this review doesn't directly outline a specific frequency at which recruitment maneuvers should be performed, it does allude to the idea that they are done intermittently throughout a (hospital) shift or once per day
[315] Metz, 2016b - Another video showing a version of the stepwise recruitment maneuver on a set of lungs attached to a vent circuit
[316] Hess, 2015 - This paper outlines these types of stepwise recruitment maneuvers and has a graphic to show how they increase usable volume in the lungs over time

that seeks to identify the point of maximal benefit by observing SpO_2 as PEEP is titrated back down from the point of maximum pressure.[317] This is particularly important to keep in mind, as the benefit of a recruitment maneuver (i.e., the involvement of more alveoli) may not be sustained if we subsequently revert back to a PEEP that we were at before the maneuver.

In any event, the utility of recruitment maneuvers is to get more alveoli involved in ventilation. This improves compliance and allows us to ventilate to our tidal volume goal with lower driving pressure while working to correct ventilation/ perfusion (V/Q) mismatch across the lung and improve oxygenation.[318] While there are risks involved and the data is a bit vague when it comes to long-term benefits, it seems fair to conclude that if we mitigate those risks by using a stepwise approach and monitoring for patient decompensation along the way there is likely some use in the transport setting.

[317] Claire & friends, 2019 - See this page for a step-by-step description of how to perform the Staircase Recruitment Maneuver
[318] Hartland & friends, 2015 - We cited this study back when we discussed absorption atelectasis in **Oxygenation**; while it looks at a specific group of patients we don't often encounter in transport (those undergoing abdominal surgery), the findings support the idea than recruitment maneuvers are of benefit in the short term (which is likely comparable to the transport setting)

Make a (Calculated and Informed) Plan

This next section covers how we go about setting the patient up on the ventilator. In particular, it looks at how the process differs when it's us initiating ventilation versus if we are taking over a patient in which ventilation has already been initiated. This may not seem like a big deal, but the taking over of a ventilated patient is a bit tricky. Even though we have these predetermined strategies for different patient types, the truth is that there is a lot of variation in how patients respond to the vent. Sometimes an asthmatic patient is happy with an I:E ratio of 1:2, other times a hypotensive patient has a high respiratory rate and low tidal volume for good reason, etc. Because of this, we need a method to determine when changes are needed and when we can leave things alone as we find them.

Getting the Intel Ready

The first thing we do for any patient who needs to be or is already ventilated is to listen. We listen to a report from whoever was hanging out with the patient before we got there. This is very important for all patients, as it can tell us how the patient has responded to or will respond to strategies we might have in mind. We then (as in *after* listening) decide on a strategy based on how we think that patient ought to be ventilated. Next we get a reasonably accurate patient height (either from a reliable healthcare provider, by measuring it ourselves or looking at the patient in the flesh and making an estimate) and perform three calculations: **Ideal Body Weight**, **Tidal Volume**, **Minute Volume**.

Following that is the patient exam. We'll discuss a few of the specifics when we talk about a patient already on the vent, but we for sure want to get an exam done before we start manipulating things or playing with our vent. Our mental construct of a strategy based on the report we received should match what we see in the exam. If not, we need to clarify that amongst ourselves before moving forward. No need to elaborate on that here, we all know the importance of a good assessment.

And then we might also, at this point, need to consider specific data points that might dictate or direct vent strategy:[319]

	presentation	vitals	labs	trends
obstruction	respiratory distress, wheezing, cyanosis, hives	high RR	elevated $PaCO_2$	decreasing respiratory rate and/ or effort (fatigue)
hypotension	pale or cool skin, significant bleeding, obviously sunken fontanelles	low BP	low H&H, high lactate	decreasing BP, decreasing LOC
acidosis	Kussmaul's respiration	high RR	low pH, high lactate	decreasing respiratory rate and/ or effort (fatigue)
lung injury	difficulty breathing	low SpO_2	low PO_2	increasing oxygen requirements

Note that this is not meant to be exhaustive, rather just a reminder to consider findings to direct vent strategy in various locations.

So once we have a report, have done an assessment, and are decided on a strategy, we move forward.

[319] We'll discuss **Blood Gas Analysis** later, but for the time being we can simply look at a few specific data points

From Scratch

When we are the ones initiating the vent, it's fairly straightforward: we take the settings we've come up with based on presentation and pathophysiology, then plug them into whatever mode and method of control we decide to use. We've already talked about the different strategies and why we may choose to use one mode or control over another, so we won't spend any more time on that here. The easiest way to do this is to stick with whatever our machine defaults to and then adjust from there if need be. For guidance on choosing other initial settings, refer back to **A General Vent Strategy** or any of the **Specific Vent Strategies** as applicable.

And then to review situations in which we might opt to deviate from default settings on the vent and preferentially choose one of the specific **Modes of Ventilation** or **Control of Ventilation**, consider the following:

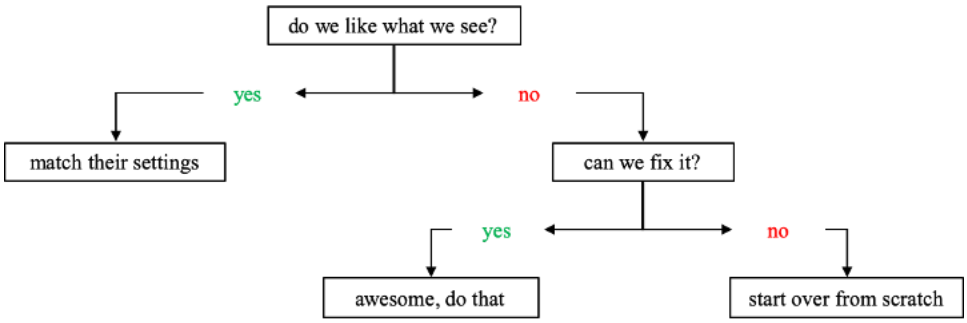
when to consider deviating from default mode & control			
mode of ventilation		***control of ventilation***	
assist control	this should be and probably is the default mode, but consider specifically with **hypotension** and **acidosis**	***volume***	**obstruction**
SIMV	significant concern for AutoPEEP that cannot be managed otherwise (unlikely scenario)	***pressure***	risk of pneumothorax

Once the patient is on the ventilator, we just need to confirm that everything is going as planned, beginning with the **Three Big Things**: oxygenation, ventilation, and comfort. Once we get those things sorted, we can then move on to some of the finer subjects (which will be discussed in the next section, **Keeping Things Going**).

Just to reiterate: the settings we conceptualize prior to initiating ventilation (and as discussed in the previous sections) are starting points from which we then make adjustments. It may very well turn out that we end up with settings, based on patient need, that vary significantly from what we initially had in mind and that's OK. But the starting point ought to be based on an understanding of what is going on with the patient and calculated goals. And if we have no idea which strategy to choose or if the patient fits too many categories all at once, just start with those basic settings we discussed in **A General Vent Strategy** and go from there.

Patient Already on the Vent

With someone already on the vent, it gets a little more complicated. We'll draw it out in a short, simple algorithm first and then we will expand on it as we go:

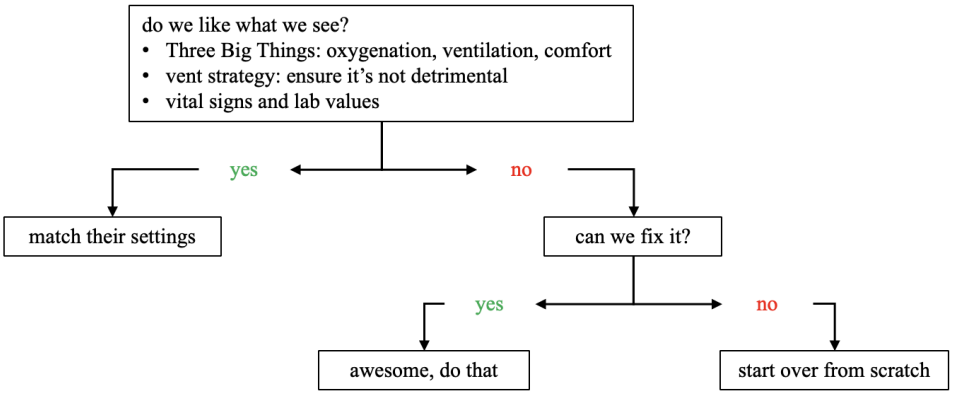


The first step in this little algorithm, "do we like what we see?" refers to a few different things: First of all are the **Three Big Things**: oxygenation, ventilation, and comfort – those for sure need to be addressed. Second is strategy: are the chosen settings at odds with what we had in mind? In the case of a hypovolemic patient with a high respiratory rate, for example, we may say, "yes, this strategy may be detrimental to the patient." In the case of an asthmatic patient with an I:E ratio of 1:3 we may decide, "this isn't what I would've set up from scratch, but let's see if it is working for the patient or not before deciding to change things." The idea here is to see what puts our patient at risk and what doesn't: a high percentage of time at decreased preload (%TaDP) and hypotension does put a patient at risk, while an I:E ratio of 1:3 in an asthmatic with no AutoPEEP doesn't.[320]

So we addressed the Three Big Things, we made sure the existing strategy isn't counterproductive based on what is going on with the patient, then we look at vitals and labs. The idea is to ensure that both perfusion and acid-base balance are all good, in the context of our vent strategy, and that we don't identify a life-threatening value or pattern of values with whatever information we have available. No need to get into specifics here, but if all is well in each of those general three subject areas, then there is no reason for us to go messing with settings and we should match what they are using.[321] The only exception here is if our machine can't do the settings they have. For example, if the patient is on **Pressure-Regulated Volume Control** and we don't have that choice, then we match their settings as best we can in either **Volume Control** or **Pressure Control** and go from there.

But what about checking a **Plateau Pressure** and **AutoPEEP**? If our patient is alive and well and passes an assessment in all three categories we just discussed (the Three Big Things, vent strategy, vitals and labs), then those things can wait until we get them on to our vent. Some reasons for this: the delay here is only a few minutes at most, the measurements may vary by machine (i.e., how individual breaths are delivered), and we've already determined that the patient is stable via a number of different assessment parameters.[322] And while scene time may or may not be a valid reason, we do want to use time efficiently and get patients moved unless we have reason to delay.

Let's redraw that algorithm we started with and add in just a little bit of detail to include all of these ideas. Then we'll move on to the next question and talk about it further:



Next question to discuss further is, "can we fix it?" We'd like to address whatever issues we have (as determined by our assessment in the first box of the algorithm) by way of one or two interventions and keeping the majority of settings as they are. For examples: if the patient is uncomfortable and we can provide analgesia on top of the sedation they are already getting or simply make an adjustment to fix a dyssynchrony, that may be

[320] To review these concepts: I:E ratio was in **Inspiratory Time (and I:E Ratio)**, percentage of time at decreased preload (%TaDP) was in **Hypotension**, and **AutoPEEP** in this context was in **Obstruction**

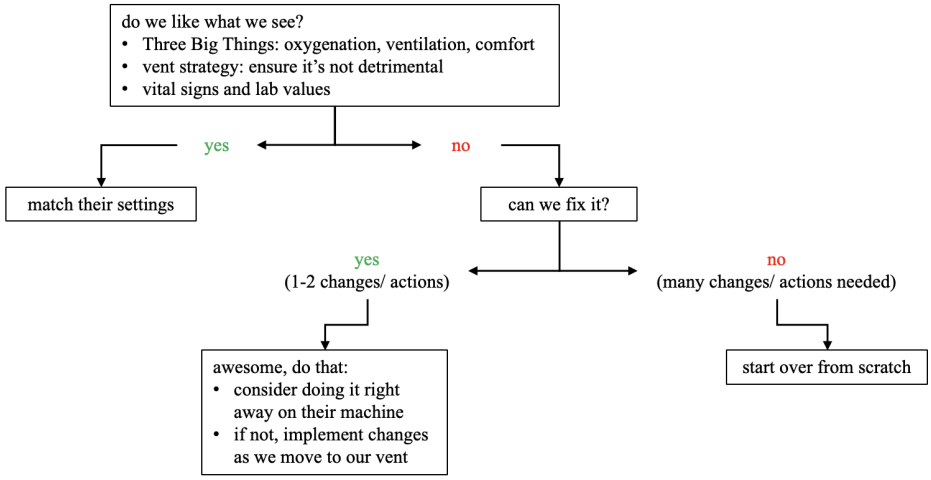
[321] And to brush up on **Blood Gas Analysis**, navigate to any of the following resources:
Woodruff, 2007 - Article that outlines a six-step approach to arterial blood gas interpretation
Smith, 2014 - Short video to review the basics of blood gas interpretation; while he uses kPa versus mmHg to outline normal values, it's the best of this type we've been able to find

[322] There's also the argument that we may not know how to manage the hospital vent or measure these things on a particular device

all that is needed; if we can fix a high $EtCO_2$ by increasing **Tidal Volume** (or **Respiratory Rate**) a bit, no need to change mode or control; if we can address a potential for hypotension by decreasing rate and then increasing tidal volume, all is good; etc.[323] If, however, we are getting into a situation where it will take lots of changes to set things right, it may make the most sense to start from scratch with a whole new set of parameters. And in that case we may as well change a bunch of things and go with our preferences.

One thing worth mentioning here is that it is sometimes cool for us to make these changes as the patient lies and on the sending facility's (or crew's) machine. Other times we make adjustments as we transition to our machine. We for sure want to avoid alienating the transferring staff by messing with their machine if that relationship doesn't exist, so just be cognizant that are two sub-options in the "awesome, do that" course of action: do it right now and on their machine or do it as we transition on to our machine. Last thing and probably already obvious is that there is some middle ground here: we may make some changes right away and then defer other things until transfer, all as part of the same strategy. Example: give sedation now, adjust tidal volume or rate during the transition.

And one more time, let's see how the algorithm would look with these additional details added in:



If at any time during this whole process things get too weird, we can always skip ahead to the "start over from scratch" end of things. Just recognize that the more changes we make, the less able we are to evaluate the efficacy of a single intervention. Like a science experiment, it helps to isolate variables and know that the observed result can be attributed to a specific adjustment. And even though we mentioned it already, interpersonal dynamics also come into play here: make changes based on necessity, not on personal preference. That will help maintain positive relationships with referring staff and crews.

[323] We discussed these things in the sections on **Comfort**, **Ventilation**, **Hypotension**, and **Asynchrony & Dyssynchrony**

Initiating Ventilation

Once we have a plan in place, the next step is to get the machine ready to go and to start doing the things we want it to. Power on the machine, connect the circuit, and perform any required safety checks. These checks could include self-tests to evaluate internal components, circuit tests to ensure the circuit is intact without leaks, and/ or button tests to make sure all of the buttons, dials, and knobs work OK. Details on that live in the manufacturer's documentation for each vent, protocols for when to do these things (i.e., at bedside vs. at start of a shift) depend on agency policy.

Next we dial in the settings we came up with in **Make a (Calculated and Informed) Plan** and when we are ready to start ventilating on our machine we attach the circuit and initiate ventilation. On many vents there is a standby option so that we can dial in settings and then leave it sit until we are ready. This allows us to prep the machine until it is time to initiate and allows us to sync the start or transfer to our vent with our patient care workflow. For example, some crews choose to do this prior to or after moving a patient onto their stretcher.

Another thing to mention is the use of a test lung to facilitate setting up the ventilator. In the past we would choose settings and initiate ventilation into a fake rubber lung (vs. the patient directly) until it was time to begin ventilating the patient. We would also see clinicians then increase high-pressure limits and decrease low minute volume alarms to avoid the machine making noise while waiting. Now that standby modes are commonplace and pre-operation checks ensure the machine will work as intended, this is no longer necessary. It is also important to mention that using a test lung in **Pressure-Regulated Volume Control** can lead to underventilation of an adult or overventilation of a small child, as the pressure (and therefore volume delivered) will adjust to the compliance of the test lung.[324] For all of these reasons, the test lung is generally not recommended with modern transport vents.

Now we attach the vent circuit to the patient's endotracheal tube or other advanced airway,[325] we start ventilation, and then we observe to see how the patient handles the transition. The specifics of how to monitor that are discussed in the next section, **Keeping Things Going** and we outline what to do with **Acute Deterioration** just after that, but it does bear mentioning what to expect as we initiate ventilation.

Recall back to our discussion of **How is Positive-Pressure Ventilation Different?** that our primary concern when initiating positive-pressure ventilation is hypotension. While the ventilator may not be the start of positive-pressure ventilation in our treatment (i.e., maybe the patient was being ventilated manually for some time), it may very well be the case that the intrathoracic pressure was not sustained by maintaining an appropriate mask seal as consistently as it will be with mechanical ventilation.[326] So be prepared to mitigate this effect when initiating ventilation.

When transferring a patient from one vent to another, such as during an interfacility transport, it is not all that uncommon that we see a transient drop in measures of **Oxygenation** and/ or evidence of discomfort. For this reason we sometimes see clinicians initiate ventilation with an FiO_2 of 1.0 and address **Comfort** with both **Analgesics & Sedatives** prior to transfer. This is fine to do proactively or we can simply address these things as they come up. The only caveat here would be to mitigate a transient dip in SpO_2 with FiO_2 rather than **Positive End-Expiratory Pressure** at this point. FiO_2 is a quicker fix and PEEP will both take time and cause more downstream effects.[327] And then once oxygenation has stabilized, we can titrate FiO_2 back to a more appropriate level.

In any case, taking a patient off of the transport vent and putting them back on the facility's vent or ventilating them manually with a BVM is not the solution. Transport clinicians should both anticipate these events and know how to mitigate them on the vent while leaving the system intact.[328]

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[324] On the Hamilton T1 "volume control" or adaptive pressure ventilation (APV) are PRVC modes

[325] Possibly in coordination with clamping the tube, as discussed in **Lung Injury**

[326] Roveri & friends, 2025 - This is not to suggest that BVM ventilation (with or without PEEP valves) does not work, as has been demonstrated in the linked paper, simply that application in the out-of-hospital setting is much different than in a controlled experiment

[327] The speed at which FiO2 and PEEP take effect was discussed in **Oxygenation**; how to best adjust PEEP is discussed in **Optimization of PEEP**

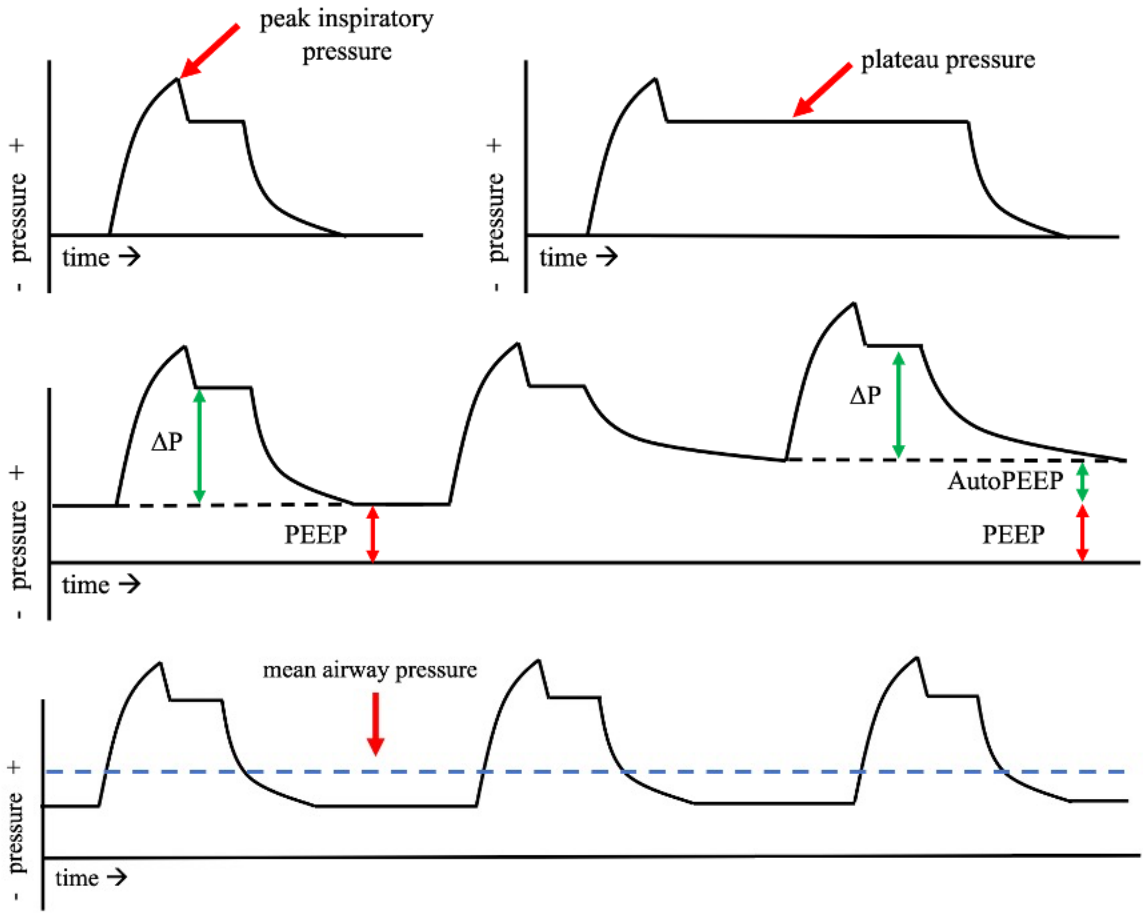
[328] This idea is discussed again in **Acute Deterioration**

Keeping Things Going

This next section goes over what we do once we have the patient on our machine and the **Three Big Things** (oxygenation, ventilation, comfort) have all been addressed. We already talked about how we sometimes vary from the settings we start out at and this section explains how that happens. We want to both avoid injury and optimize air delivery, so we make adjustments to work towards those goals.

Watching Pressures

We discussed these things already, but here they are again: **Peak Inspiratory Pressure**, **Plateau Pressure**, **AutoPEEP**, **Mean Airway Pressure**, and **Driving Pressure**. And for visualization, in case we forgot, here's what they look like on a pressure waveform in **Volume Control** ventilation:

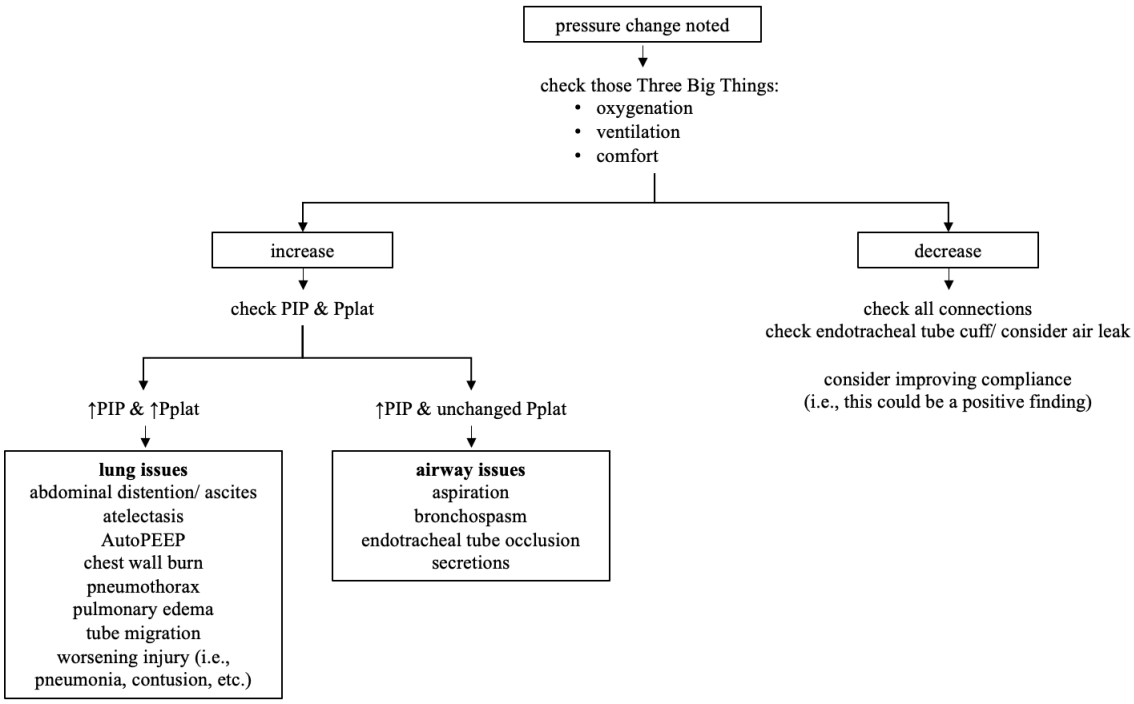


High for peak inspiratory pressure is 35cmH_2O, although we may go beyond that in certain situations (such as a small endotracheal tube). Plateau pressure max is normally 30cmH_2O and we do try to stick by that one whenever possible except in those cases where plateau pressure may not reflect alveolar pressure.[329] AutoPEEP is normally zero; we generally take actions to address AutoPEEP when we see evidence of it, but may tolerate a small amount before doing so. As for mean airway pressure, we don't generally cite a normal range, but know that a change in this value can be the first indicator of an alteration somewhere in the system. And driving pressure is most relevant with **Lung Injury**; in those cases we try to limit it to 15cmH_2O. All of

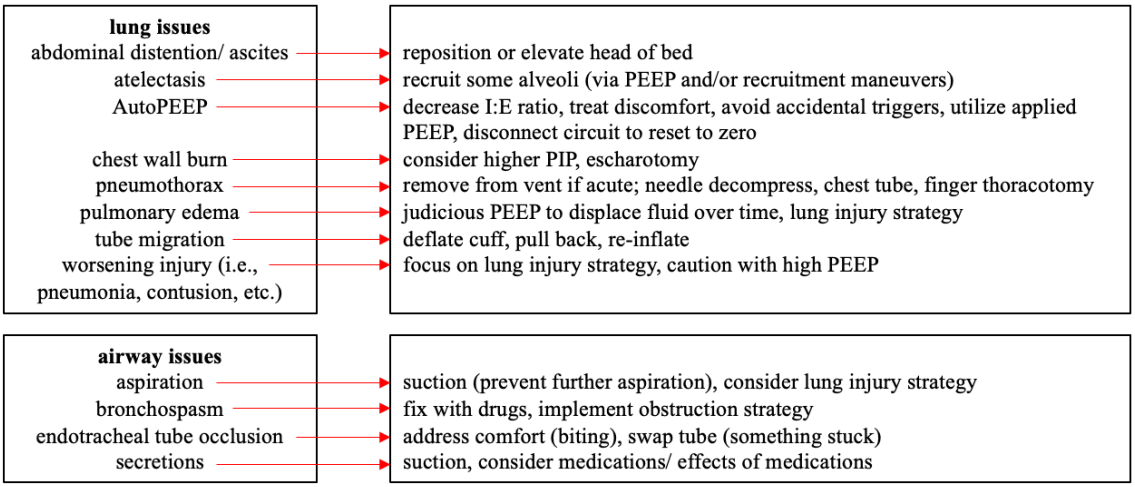
[329] We talked about these situations in **Plateau Pressure**, but just to reiterate: we assume plateau pressure is a reflection of alveolar pressure, but things that impede lung expansion can also give a high value

these parameters should be checked (when possible, depending on control and patient's respiratory effort) within the first few minutes after placing someone on our machine and then again periodically through transport.[330] It may help to simply add these pressures on to a mental list of things to reassess as we go.

As far as what to do with this information once we have it, here's a flowchart to help sift through the information and take action to address potential problems:[331]



And then let's look at potential solutions for each of these cases:[332] [333]



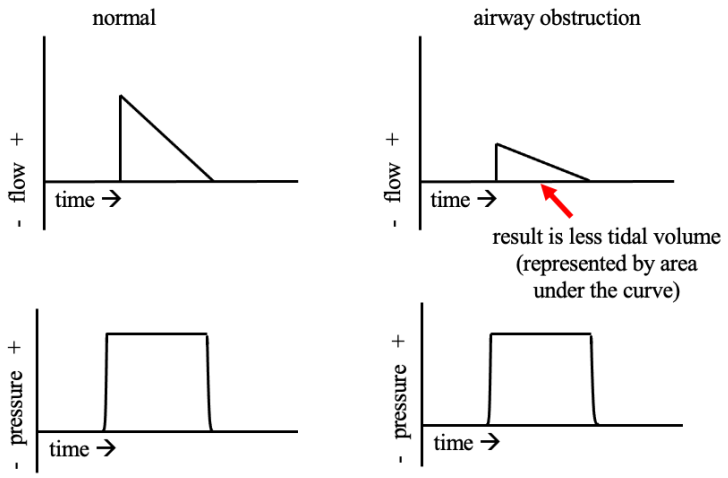
[330] For example, if a patient is triggering lots of breaths, we may not be able to get an AutoPEEP/ do an expiratory hold; if they are in pressure control ventilation, we may not be able to do an inspiratory hold (due to limitations of a particular machine)

[331] Lodeserto, 2018 - The left bit of this chart is similar to one he puts forth in his series on vent management

[332] Briggs & Freese, 2018 - There are also lots of weird cases out there to explain things that can happen, the chart above should not be assumed to be an exhaustive list of causes or fixes; as an example, this referenced article outlines a case of high airway pressures related to an endotracheal tube positioned with the bevel up against the wall of the trachea - the fix here was simply to rotate the tube 90 degrees

[333] And to link back to sections listed in this graphic: **Positive End-Expiratory Pressure**, **Inspiratory Time (and I:E Ratio)**, **Volume Control**, **Lung Injury**, **Comfort**, and **Obstruction**

In **Pressure Control** ventilation when we may not have access to plateau pressure to identify these trends, there are other parameters we can look at. Most obvious is exhaled tidal volume: as compliance decreases, exhaled tidal volumes will drop (and vice versa).[334] In the case of airway obstruction, oftentimes we won't notice initially because the machine essentially accommodates for this increased airway resistance by using less flow:



Since we don't always monitor waveforms with transport ventilators, an airway obstruction may not get noticed in pressure control ventilation until it is severe enough to impact exhaled minute volume.[335] The best way to catch these sorts of things before they have an impact on patient outcome is by setting alarms appropriately so that we are notified right away as things change (see following section).

Alarms[336]

Next on our list of things to discuss are alarms. We won't talk about all the alarms that our machines might have, but we will talk about a few of the important ones. Alarms can be broken down into two general categories: ones that are default on the machine and ones that are set or chosen. Those default ones may be different between machines, but deliver similar messages like, "hey friend, our circuit got disconnected" and "oh snap, we ran out of oxygen." Those ones can be referenced and learned about in the manual for whatever machine we happen to be using. The other ones, the ones that we set, are the ones we'll focus on here.

One important alarm we set on the machine is the high-pressure alarm (which goes off when our high-pressure limit is reached). The reason this alarm is so important is that if it gets triggered, inspiration cycles off. That means that if we have a situation where we repeatedly trigger a high-pressure alarm, we may end up with a **Minute Volume** that bottoms out and a patient that quickly deteriorates. Imagine we place a patient on the vent who has either an untreated airway obstruction or poor compliance. If we try to ventilate this patient in **Volume Control** and at normal settings, every breath that goes in might trigger the high-pressure

[334] And to review this idea of monitoring compliance in pressure control ventilation, refer back to **Compliance & Resistance**

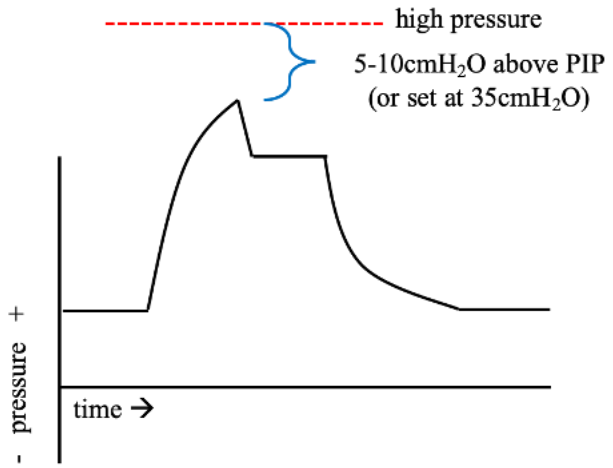
[335] We can also (again, this is in pressure control) look at flow as calculated and delivered automatically by the vent; higher flows mean less resistance, so even if we don't know ranges or normal values we can still use this concept to trend changes; also look to **Vent Graphics** and **Waveforms** for guidance on these ideas

[336] Disclaimer about this section: there isn't much out there in the universe to provide guidance on how we should set these alarms; there are studies that have collected data on alarm settings for in-patient units, but we don't feel it would be appropriate to apply those to the transport setting; given that we move these patients one at a time with one or two providers (versus a unit full of ventilated patients, lots of alarms at once, and higher patient ratios) we should arguably always have eyes on the machine and it makes sense to use much tighter limits for alarms than we might see in the hospital setting; that said, this is just one opinion on the whole thing

alarm and get terminated early with a net result of almost no minute volume. The reason this safeguard exists, in spite of this risk, is because we could for sure cause a lot of damage if we accidentally give too much pressure.[337]

Moral of the story here: if we are in volume control ventilation and have a concern for increased airway pressures, we should consider going up on the high-pressure limit before putting the patient on the machine to avoid dropping our minute volume. On the flip side, in **Pressure Control** we need to vigilantly monitor exhaled minute volume (and also exhaled tidal volume) to avoid the same issue (of decreased minute volume). This leads us to the next most important alarm we can set: low minute volume. We set this limit at a reasonable value below our minute volume goal so that if things get weird and minute volume starts to drop, we get notified right away before our patient suffers. In this way, we utilize the high-pressure and low minute volume alarms to simultaneously ensure both safety and adequate ventilation for our patients.

As far as setting the high-pressure and low minute volume alarms, that is a bit dependent on our margin of safety and when we want to be notified of changes in the system. As a general rule of thumb, the high-pressure limit should be no more than 10cmH_2O above our **Peak Inspiratory Pressure**. If, however, our peak pressure is already high of normal, consider setting the high-pressure alarm 5cmH_2O over that value or at our upper limit of 35cmH_2O:



In the event of one of those situations which may lead to repeated triggering of the high-pressure alarm and sudden drop in minute volume, increase the high-pressure limit (even beyond 35cmH_2O if need be) to maintain minute volume. Note that this would be a short-term fix and we should start to consider other strategies right away: trial pressure control, consider pharmacologic and procedural interventions, etc.[338]

As for the low minute volume alarm: set that within 25% of the minute volume goal that we calculated when we first started into this process of getting the patient on the vent.[339] If we have a patient breathing in excess of that goal and we want to know if that changes, we just set the low minute volume alarm 25% below what they are currently at. In any case, the low minute volume alarm is just a catch to alert us when we've missed a change. Typically we will be on top of these trends and notice things before the alarm even gets sounded, but sometimes we get distracted by other interventions and this backup system can keep us informed.

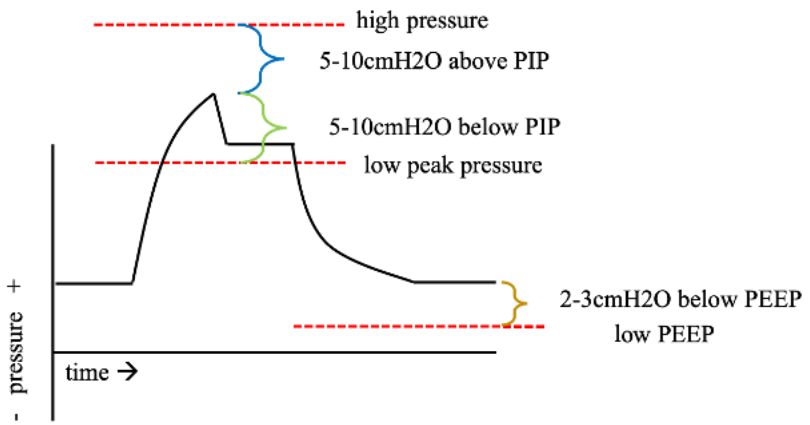
Other alarms that we can set to help us better keep track of what's going on with the vent and our patient are low peak-pressure, low frequency, high frequency, and low PEEP. Low peak-pressure alerts us when the peak inspiratory pressure is lower than we would expect; this could indicate a cuff leak, increase in patient's respiratory effort (i.e., negative pressure produced with patient effort), or a loose connection (an actual disconnection would probably trigger a disconnect alarm, one of those non-adjustable alarms consistent across machines, as the pressure would drop much more significantly). Low frequency can let us know if the patient's

[337] This is illustrated in the section on **AutoPEEP**
[338] See **Medications in Mechanical Ventilation** and **Acute Deterioration** for further discussion
[339] And this 25% figure for where to set the low minute volume alarm is an arbitrary number that we feel is appropriate, there aren't too many specific recommendations for this type of thing

Respiratory Rate starts to decrease. This is good if the patient is consistently breathing above a set rate and we want to be aware if that intrinsic effort changes. And reasonably enough, the high-frequency alarm advises us when the patient starts to breathe faster or if some mishap is causing the machine to think that (s)he is. Lastly, low PEEP lets us know if the end-expiratory pressure drops below our set **Positive End-Expiratory Pressure**. This could indicate a leak, cuff deflation, or even an uncuffed tube (with pediatrics) that is too small.[340]

That's just a quick overview of alarms; recognize that the most important ones are high pressure and low minute volume, but that there are a number that can help us be aware of changes in the system as we work through a transport. Because there is so much variation between machines, the best way to get familiar with the alarms we will be working with is to read the manual that comes with the machine. Super fun reading, but it's good information and can help us fine-tune the feedback from the vent so that we can better monitor what's going on with the patient.

And we'll end with a graphic to show how some of these alarms would be represented on that pressure over time waveform in volume control ventilation:



Optimization of PEEP

Positive End-Expiratory Pressure (PEEP) optimization includes a few different things: choosing a reasonable initial setting,[341] determining which patients might benefit from subsequent changes, understanding how frequently we should be adjusting the thing, and then making changes and evaluating their efficacy quantitatively. Let's work through each of these in turn.

We mentioned before that PEEP at a default of 5-6cmH_2O is just fine for most patients. We've since discussed that we may choose to go lower than that in a **Hypotension** vent strategy. That's fine to do if we don't have a better way to address hypotension or even temporarily while we make those things happen, just keep an eye on indicators of **Oxygenation** and recognize that a normal SpO_2 in the setting of anemia (i.e., hemorrhagic shock) may leave the patient in a deficit.

[340] Also discussed shortly in **Acute Deterioration**
[341] This concept was already mentioned, but we'll include a discussion here so that all the PEEP things live in one place

On the other hand, if we have a known hypoxic state or some sort of pathology in which we expect gas diffusion at the alveoli to be inhibited to some degree, we may choose to start with a higher PEEP. We can make this simple and go with 10cmH_2O in these patients[342] or we could refer back to the "lower PEEP/ higher FiO2" chart from the ARDSNet titration protocols and match our PEEP to whatever FiO2 we think may be necessary:[343]

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

And then moving on from here, PEEP can be increased to facilitate **Oxygenation** and/ or **Ventilation** and should be decreased to avoid barotrauma and/ or volutrauma. In terms of oxygenation, recall that PEEP is just one way to address the issue and that increasing FiO_2 is a bit quicker of a fix and with fewer consequences when used short-term. That said, if SpO_2 remains low with an FiO_2 of 1.0, PEEP is the next step. As for ventilation, recruitment of alveoli with increased PEEP facilitates gas exchange in the alveoli. If a goal **Minute Volume** or desired $EtCO_2$/ $PaCO_2$ cannot be achieved, consider increasing PEEP. Just recall that sometimes it's AOK to allow for a state of permissive hypercapnia and, therefore, it is not a set rule to increase PEEP for all cases in which ventilation is below goal.[344]

With concern for barotrauma and/ or volutrauma, the first line intervention should be to keep **Peak Inspiratory Pressure** and **Plateau Pressure** within safe limits (<35cmH_2O and <30cmH_2O, respectively). This is typically done by adjusting either **Tidal Volume** or the **Pressure Control** parameter. If PEEP started out high or was increased and it's not possible to maintain ventilation with a lower tidal volume (or pressure control value), it may be necessary to decrease PEEP. That said, this is not all that common. If we do end up with an elevated PEEP (such as after matching settings from another machine) and are concerned for overdistention, we can simply decrease PEEP and evaluate if that made things better or not - we can always go back up if need be.

Alright. So when to do these things and how frequently? There is no straight answer here, but it is worth noting that these things often take time, however that isn't always the case. Some experts recommend letting a patient ride initial or new vent settings for thirty minutes before considering another change, others will stand at the bedside and make changes one after the other until a target outcome is achieved. When adjusting PEEP, we recommend waiting *at least* five minutes and recognize that some outcomes may not manifest in the time it takes to complete a normal transport.[345]

And as for how much to change PEEP at a given time: 2-3cmH_2O is just fine. Less than that may not have the intended effect we are looking for and more than that can cause negative consequences if we overshoot our goal. That said, there's a bit of nuance here. A change from 5cmH_2O to 10cmH_2O feels a bit less drastic (and more acceptable) than a change from 10cmH_2O to 15cmH_2O. Likewise an increase by 5cmH_2O feels more significant than a decrease in 5cmH_2O. Again, it's a bit of a grey area.

Evaluating the efficacy of a change depends, in part, on our initial goal. If we increase PEEP to facilitate ventilation (for example: VE below goal and $EtCO_2$ 50, so PEEP increased to 12cmH_2O) and subsequently see our previously-elevated $EtCO_2$ decrease towards a normal range, all is well. Same goes if we decreased PEEP for high pressures and now they are lower and safer. That said, there are other techniques for this that are more applicable across the board.[346] The best way is to look at compliance. If we make a change to

[342] Bishop, 2022 - This is similar to the recommendation made by @MelodyBishopRT in her free online vent book
[343] NHLBI ARDS Network, 2004 - This was first mentioned and cited in the section on **Lung Injury**
[344] Pruitt, 2007 - Permissive hypercapnia discussed here, also in both **Obstruction** and **Lung Injury**
[345] Chiumello & friends, 2013 - These guys did a very cool study in which they made changes to PEEP, both up and then down as well as down and then up, and measured how long it takes for changes in oxygenation, ventilation and lung dynamics to equalize, times varied from five minutes to over an hour depending on both the flavor and direction of change
[346] Hess, 2015; Edginton & friends, 2024 - We cited the first paper in **Recruitment Maneuvers**, it includes an overview of different techniques to assess appropriate PEEP; the second one is a scoping review of all the different techniques

PEEP in either direction and compliance gets better (i.e., we see a bigger number), then the change was effective.[347]

Alternatively we could look at **Driving Pressure** as an approximation of compliance. In **Volume Control** we see a decreased driving pressure with increased compliance. So if we change PEEP and driving pressure goes down, all good and the change was beneficial. In **Pressure Control** the idea is similar, but since driving pressure is fixed we instead look to exhaled **Tidal Volume** (Vte) - if we change PEEP and Vte increases, then compliance got better and the change was beneficial.

Other techniques include evaluating **Oxygenation** (commonly via SpO_2 in transport, but maybe also by **Blood Gas Analysis**), comparing changes to an FiO2/ PEEP table (similar to the one shown above), considering **Vent Graphics** (specifically the pressure-volume loop), lung ultrasound (manually assessing for improved aeration) or correlating with the **Expiratory Time Constant**.[348] That said, compliance is best, **Driving Pressure** is a decent second, these others are OK. And as with all assessment tools, utilization of more than one strategy to confirm findings is always fine to do.

Titrating Up on Tidal Volume?

Up to this point we've recommended considering **Tidal Volume** above that 6-8ml/kg range in just a few circumstances: to increase minute volume (in the **Ventilation** section), with airway **Obstruction**, and as part of the **Hypotension** strategy. But along the way we've mentioned that **Dead Space** is less than ideal and that more tidal volume is preferred to a higher respiratory rate. We also said that more breaths over an arbitrary amount of time means more of both that percentage of time at decreased preload (%TaDP) concept and stress on the system. And then there is also some data that lower tidal volumes predispose ventilated patients to **Asynchrony & Dyssynchrony**.[349] Given all of these things, why would we not advocate for a higher tidal volume, lower respiratory rate strategy in all patients? The answer to that question isn't completely clear, but we'll try and answer it as best we can.

First thing to realize is that much of what we know about vent management these days is based on the ARDSNet study that compared a tidal volume of 6ml/kg to one of 12ml/kg with pretty significant findings.[350] Since then, most of the research out there has focused on this lung-protective approach. While there have been a few studies that looked at this middle ground of 8-10ml/kg, there's been no clear benefit over the 6ml/kg approach, so the default response is that we should stick with what we know works.[351] Which makes sense, but it doesn't consider the fact that there are multiple methods by which we can ensure safety while using more volume. For argument's sake, let's outline all of the things we can do to mitigate the risks of a larger tidal volume and then we'll get back to this discussion.

First thing is that we want to limit our **Plateau Pressure** to a safe level below 30cmH_2O whenever possible, which includes when we decide to go up on tidal volume. Another concept we discussed was **Driving Pressure**. We mentioned in that section that most of the data on driving pressure is specific to the ARDS population, but also noted that there may be a case for using the value to guide treatment for patients in general. So let's just say that we want to limit driving pressure to 15cmH_2O as we outlined for the **Lung Injury** patients. Another component to consider, which we discussed in **Recruitment Maneuvers**, is that we prefer to make these types of changes in small increments and with as much time as possible between titrations. And lastly, we can utilize compliance to help guide us towards our goal.[352]

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[347] See **Compliance & Resistance** for more

[348] Edginton & friends, 2024 - All of these techniques (plus a bunch more that aren't really applicable in transport) are reviewed in this paper; the exception is the **Expiratory Time Constant** which is discussed later

[349] Figueroa-Casas & Montoya, 2016 - More on that in this paper

[350] The ARDS Network, 2000 - We cited this previously in the intro to **Specific Vent Strategies** and also in **Lung Injury**; this study is what led us all down the path of lung-protective ventilation in the first place

[351] Writing Group for the PReVENT Investigators, 2018 - This is one such study that attempted to investigate an intermediate tidal volume range; we previously cited a review of this article, but here it is as published

[352] Refer back to **Compliance & Resistance** to review this idea

In **Volume Control** we could increase tidal volume until we notice a spike in **Plateau Pressure** or a decrease in compliance. In pressure control we increase pressure until we see a decrease in compliance or no increase in exhaled tidal volume after the adjustment. Once we hit either of these limits, we then titrate back the last increase (of tidal volume or pressure control) to where things were just before the previous adjustment. To map it all out with steps in the chart representing reassessment during transport:

volume control example				
step #	**tidal volume (ml)**	**plateau pressure (cmH_2O)**	**compliance (ml/cmH_2O)**	**action**
1	500	15	50	increase tidal volume
2	525	16	48	increase tidal volume
3	550	16	50	increase tidal volume
4	575	21	36	decrease tidal volume
5	550	16	50	no change, monitor
6	550	14	61	increase tidal volume

Note that even though plateau pressure doesn't get up to our previously established limit of $30cmH_2O$, we recognize that an increase beyond a tidal volume of 550 (line 4) gave us a spike in plateau pressure and drop in compliance. Therefore we may titrate back a smidge and wait for the lungs to fill more before moving back up (line 6).

And as for a how it looks in pressure control:

pressure control example				
step #	**pressure control (cmH_2O)**	**exhaled tidal volume (ml)**	**compliance (ml/cmH_2O)**	**action**
1	10	500	50	increase pressure control
2	11	550	50	increase pressure control
3	12	550	46	increase pressure control (or stay)
4	13	550	42	decrease pressure control
5	12	550	46	no change, monitor
6	12	600	50	increase pressure control

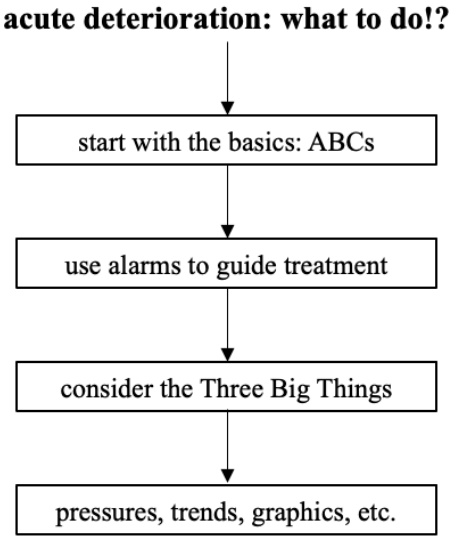
Exhaled tidal volume and compliance will likely vary from breath to breath and therefore it isn't quite as easy to recognize these trends in real-time, but the general idea holds true.

To get back to our original argument about titrating up on tidal volume: recognizing the fact that going up on tidal volume would allow us to go down on respiratory rate with the result of less dead space and less stress into the system, it intuitively makes sense to consider how we can go about making this happen in a manner that is as safe as possible. Firstly we will want to limit plateau pressure to less than $30cmH_2O$. Next we may want to limit driving pressure to $15cmH_2O$ even though that recommendation is normally reserved for ARDS patients. Beyond that we can make changes to tidal volume in small and incremental steps while simultaneously monitoring changes to plateau pressure and/ or compliance with each adjustment.

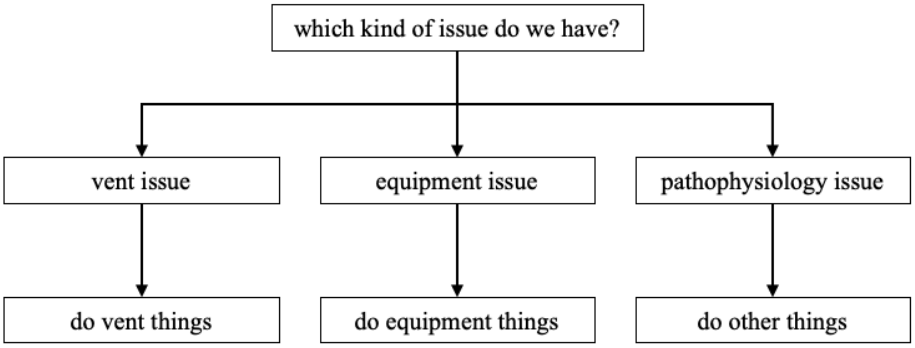
Given that all of these precautions to ensure safety are in place, we propose that going up on tidal volume is probably just fine for our ventilated patients once all the other issues have been worked through. That said, there are a few more things to mention. First is that this process takes time and many transports will exclude this level of detail in ventilator management. Second is that upper limits to plateau pressure and driving pressure are not rigid guidelines, rather they indicate endpoints at which risk clearly outweighs benefit. To say it another way, just because a plateau pressure is 28 or a driving pressure is 13, both below established upper limits, doesn't mean that it is as comparably safe as having a plateau pressure of 23 or a driving pressure of 11. And last thing to mention: there is no evidence that going up on tidal volume in this manner would benefit or improve patient outcomes, it's just something to think about and consider at this point.

Acute Deterioration

The next thing to chat about is what to do if the patient begins to decompensate while on the vent. Let's start with a simple sequence of steps, then we'll explain how we developed that framework, as well as specific interventions for various complications. The stepwise approach we recommend for troubleshooting significant issues when managing a mechanically ventilated patient looks like this:



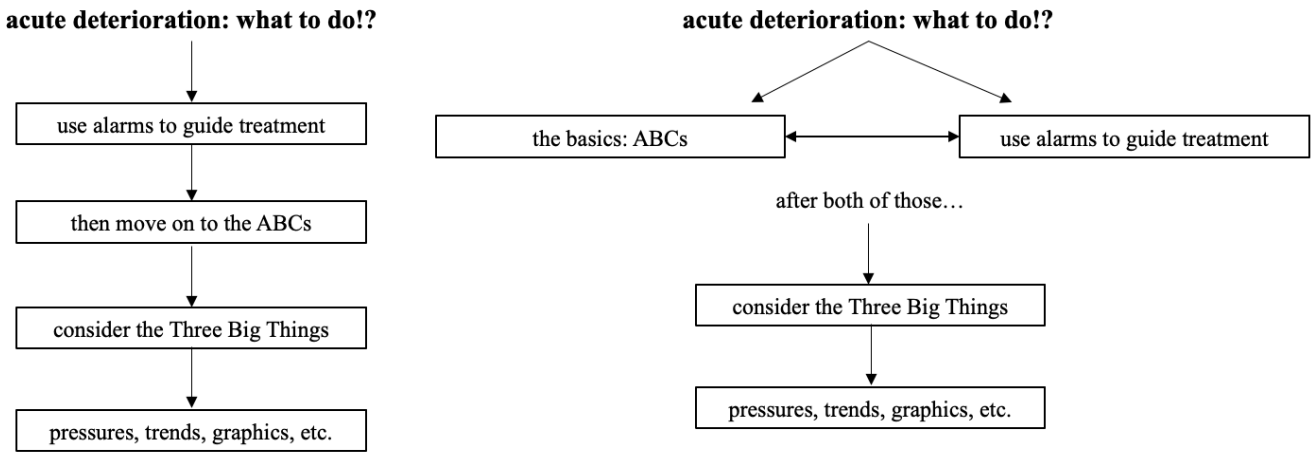
The reason for starting with a basic primary assessment is that it isn't always the vent that is the problem. And if it's not the vent, we don't need to mess with it. Think of it this way:



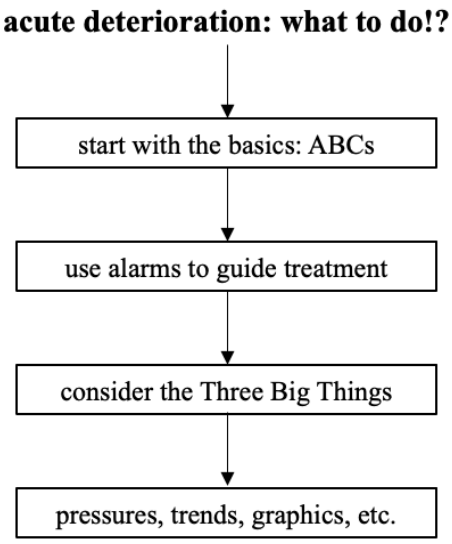
Now this is a book on mechanical ventilation, so that's the focus of this chapter, just keep in mind that we also have to manage all the other things going on with the patient and that the vent is not always the underlying reason for a patient's deteriorating status. It may also be the case that we need to do multiple types of things simultaneously. An example of this would be a patient already on the vent who experiences an allergic reaction to something. In this case we could simultaneously proceed with an **Obstruction** vent strategy and give drugs to fix the problem.[353] So while our little algorithm may be too simple, it often helps to take a moment to think about which sort of problem we have on hand and act accordingly.

[353] See **Medications in Mechanical Ventilation** for more

Given that **Alarms** sometimes point exactly to what is going on when the issue is, in fact, a vent issue, potential alternatives to the proposed framework might look like either of these:



That said, putting anything in front of the almighty ABCs is blasphemy in the realm of EMS, so we'll leave those things at the top. The important thing to note is that it is OK to quickly look at the vent alarm before jumping into action, as it can sometimes tell us exactly what the issue is.[354] So we have this as a framework for addressing acute deterioration of the mechanically ventilated patient:



Airway, breathing, circulation should be a review for anyone reading this manual, so we'll avoid getting into further detail there. **Alarms** were discussed in a previous chapter. The **Three Big Things** have been mentioned many, many times, so if there is still any doubt as to what those concepts are, refer back to prior chapters. And then lastly is to work through all the other data and feedback we have on hand, both over time and in the moment, to see if there is something we have missed. Pressures include **Peak Inspiratory Pressure**, **Plateau Pressure**, and **Mean Airway Pressure**. Trends refers to all the parameters we track on the machine itself, but also vital signs (again, don't forget the patient!). And then **Vent Graphics**, and specifically **Waveforms**, are discussed shortly.

[354] Some vents, like the Zoll EMV, actually include actionable items for troubleshooting on the display screen when an alarm is triggered

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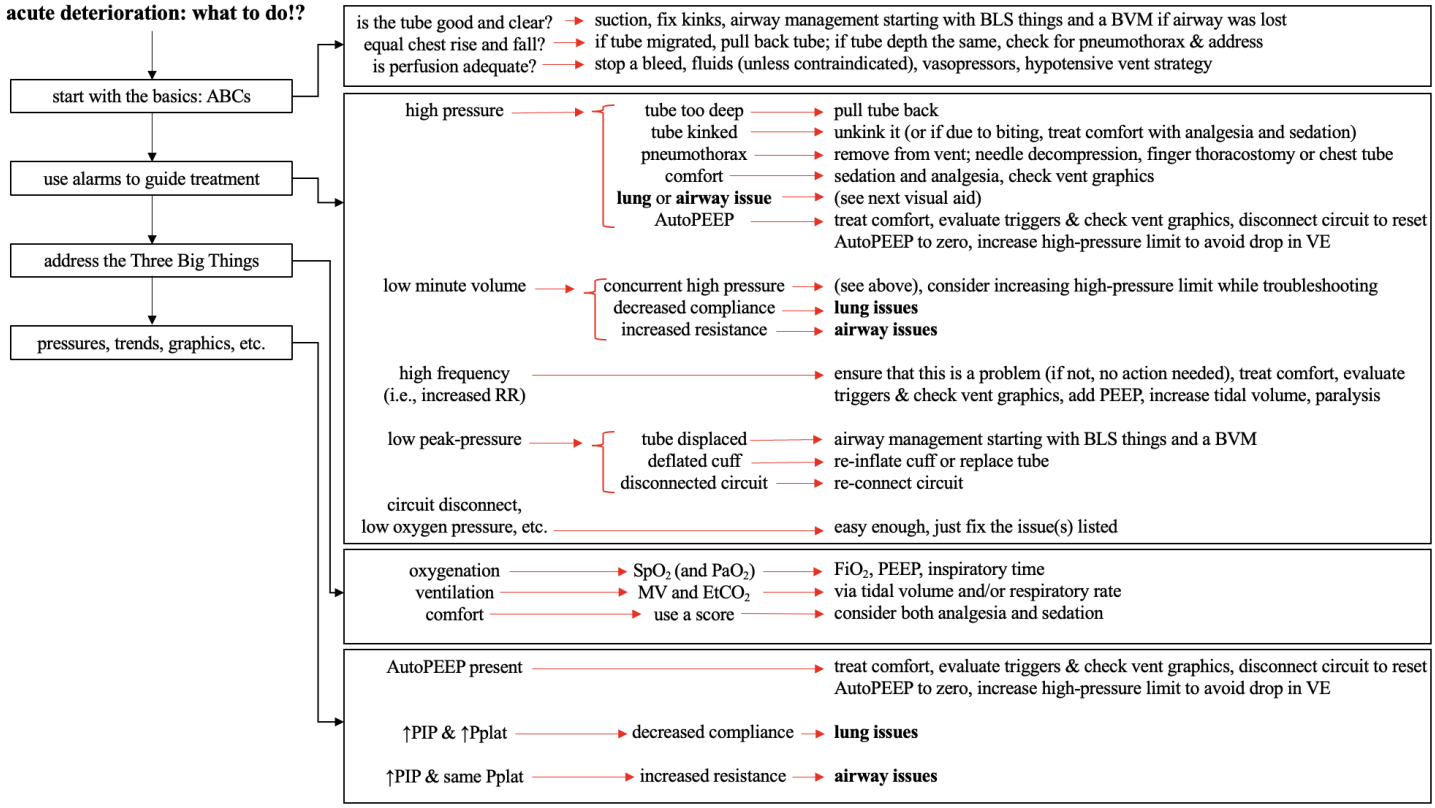
The next step is to map out the specifics for each of these four steps. It might look something like this:



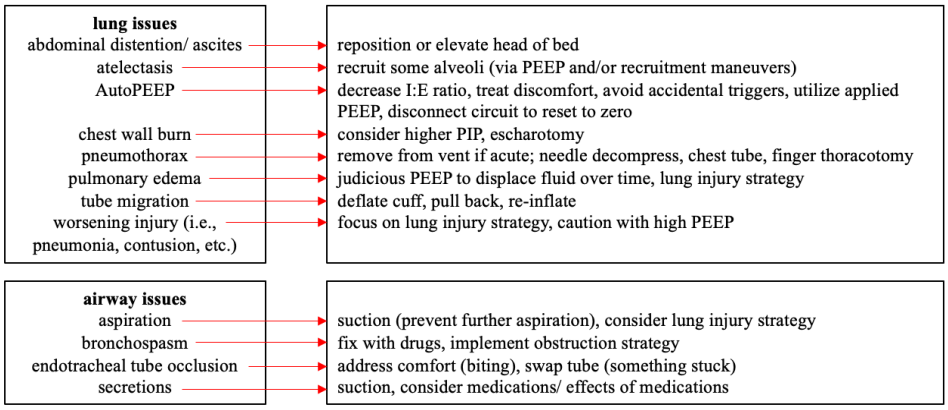
Now this may look, at first glance, like an overwhelming amount of information, but if we keep in mind that it's simply a zoomed-in or expanded explanation of four simple steps, that will help keep us on track. It's also important to mention that the things up top or higher on the list are generally more likely to correlate with acute deterioration in a patient's status. While there are definitely items towards the bottom that are life-threatening (particularly when we dig into the lung issues and airway issues), those ought to be caught in the initial troubleshooting steps. And then sometimes we miss a thing at the top and identify it further down. So there is some redundancy built into the system.

As an example, let's say we have a patient in **Pressure Control** and we note a deterioration in the patient's status due to significant secretions in the endotracheal tube with a subsequent decrease in **Minute Volume**. Ideally we'd note this right away, up in the ABCs section while assessing airway, but that's not always the case. And we may still have a good $EtCO_2$ waveform and see some chest rise and fall, indicating OK breathing. In the next box we may have **Alarms**: high-pressure or low minute volume could often, and in theory, catch this sort of thing. That said, the high-pressure alarm would not trigger in pressure control, as **Flow** gets dialed back to prevent going above the set pressure. And let's say the minute volume alarm is set inappropriately or too loose to catch the change. We move on down and finally notice that **Minute Volume** has decreased when assessing **Ventilation**.

So we address ventilation by increasing either the pressure control parameter or **Respiratory Rate**, but just for good measure we keep on working down the algorithm to see if that was, in fact, the right fix. It's in this last box that we notice a change. We see that **Peak Inspiratory Pressure** has increased, but that **Plateau Pressure** remains constant - this prompts us to consider the list of airway issues that we discussed in **Watching Pressures**. And then finally we land on our issue of increased secretions in the airway. The thing to note, however, is that even though it took a while to identify the issue, we mitigated it along the way by using a systematic approach to troubleshooting.

There's no way to accommodate all possibilities in a single algorithm without getting too crazy on the details, but that's the basic idea. Before moving on, just a few things to note. First is that a low minute volume

alarm may also accompany acute deterioration, but it will likely be tied to either a high-pressure alarm (with breaths cycling off due to that alarm getting triggered) or some kind of disconnect (which would likely be indicated by a circuit disconnect or low peak-pressure alarm). We also didn't include a low-frequency or low-PEEP alarm anywhere in this flowchart, as those probably aren't tied to an acute deterioration unless accompanied by one of these other trump cards. And then we already showed this before (and recognize that not all of these are acute life threats), but just to clarify again the different lung and airway issues we might come across:



One other random point to insert regarding the low-pressure situation related to a deflated or leaky endotracheal tube cuff: with a partially deflated cuff, remove air from the cuff and then re-inflate with an appropriate amount of air. Simply adding volume can contribute to both injury from overinflation and likelihood of tube displacement.[355] Also consider using a manometer to measure cuff pressure and establish a baseline moving forward if available. And if the cuff is defective, a bougie can be used to exchange it for a new one or take steps to fix the issue temporarily.[356]

Now we would be remiss if we didn't mention that there are other approaches to this sort of thing. The most common example of which is the DOPE mnemonic:

the DOPE mnemonic		
	issue	**action**
D	displaced tube	confirm tube placement
O	obstruction	suction, check for kinked endotracheal tube, consider bronchospasm
P	pneumothorax	remove patient from vent; decompress, finger thoracostomy, or chest tube
E	equipment	check all connections

There are also some variations of this, so we may see it out there with an "S" at the end for stacking (i.e., **AutoPEEP**),[357] an "R" at the end for rigidity of the chest wall (a rare complication of Fentanyl administration),[358] or even with the "P" to represent pain and/ or (Auto)PEEP.[359] It is also sometimes accompanied by another mnemonic called DOTTS which outlines actions that can be taken to fix issues identified by DOPE. Now DOTTS includes a step where we bag the patient with a bag-valve mask (BVM) and

[355] Tennyson & friends, 2016 - This paper reviews the incidence of endotracheal tube cuff overinflation in the helicopter EMS setting
[356] Mellick, 2014; Lauria & friends, 2019 - The first is a video that demonstrates exchanging a tube using a bougie; the second is a short article that outlines strategies for salvaging a damaged endotracheal tube
[357] Rezaie, 2018 - Describes the "S" added to the DOPE mnemonic; also gives on overview of the DOTTS idea discussed below
[358] Thomas & Abraham, 2018 - While the "R" component to the DOPE mnemonic isn't all that common, it may be worth keeping in mind
[359] Wright, 2014 - A great read on ARDS in general, but specific to this cause he's got a nice DOPE graphic that he adapted from another source

we've crossed that step out. We don't recommend routinely taking someone off the vent unless we have good reason to and we'll get back to this idea in just a little bit. But just so we can see it in its true representation, here it is:[360]

the DOTTS mnemonic		
	action	**explanation**
D	disconnect the vent circuit	to fix AutoPEEP or decreased preload (i.e., pneumothorax or hypotension)
~~**O**~~	~~O_2 100% via BVM~~	~~to manually assess for issues (i.e., look, listen, feel)~~
T	tube position or function	includes assessing placement and suctioning
T	tweak vent	consider decreasing rate, tidal volume, or inspiratory time (i.e., with AutoPEEP or hypotension)
S	sonography	consider ultrasound to identify issues (if we have it)

The DOPE mnemonic (with or without DOTTS) is easy to remember and is often cited as a guide to the initial troubleshooting process when the vented patient starts to deteriorate.[361] The concern, however, is that it is not comprehensive and does not prioritize assessments in order of importance. That said, it is used by many clinicians and is commonly referenced in critical care transport education.

With that in mind, let's summarize what actions to take in the event of an acutely deteriorating patient on the vent. We suggest a simple sequence of four steps to work through it all: check the ABCs, look at and address any alarms, review the **Three Big Things**, then check the other things (pressures, trends, graphics, etc.). If by then we haven't figured out our problem, we can consider taking the patient off the vent and bagging by hand (still not a great strategy though…) or getting out the ultrasound machine to try and identify an issue (if available).[362] While there is a well-known memory tool (the DOPE mnemonic) to guide us through troubleshooting potential issues, that tool doesn't consider feedback from the machine (i.e., alarms) nor does it prioritize assessments in a systematic way based on level of priority.

The final idea here is what to do if the patient goes into cardiac arrest while on the vent. Standard practice in this situation is to take the patient off the vent and have someone ventilate by hand while CPR is initiated. That may be a valid option if we have extra hands, but in transport with only two clinicians it may not be possible. If our particular machine allows us to ventilate without patient triggers (i.e., in controlled mechanical ventilation or intermittent mandatory ventilation), that would be the preferred approach. If not, then we may be able to approximate those **Basic Modes of Ventilation** by maxing out the sensitivity of our trigger. The reason we turn off the mechanism for patient-triggered breaths is that the machine will likely be triggered to deliver a breath with each chest compression given.[363]

Another consideration in this cardiac arrest situation is that we may need to increase our high-pressure limit so that breaths don't get cut short early during this time. If the machine is trying to give a breath while we are trying to simultaneously give compressions, we will most definitely exceed a normally-set high-pressure limit with the result that breaths will get cycled off. Ideally we could time our compressions with the machine's breaths to avoid this, but that would be difficult to accomplish. Increasing the high-pressure limit does predispose the patient to both barotrauma and increased intrathoracic pressure, but it may be the best course of action when working a code with limited resources.

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[360] To link back to sections mentioned in this chart: **Hypotension**, **Respiratory Rate**, **Tidal Volume**, and **Inspiratory Time (and I:E Ratio)**

[361] Weingart, 2011 - For some trivia on where the DOPE mnemonic came from, take a look here

[362] Mojoli, 2017; Lichtenstein, 2016 - And for those of us who do have ultrasound or simply want to learn more, the first is a short article that discusses application in mechanical ventilation; the second is a full-length textbook on lung ultrasound

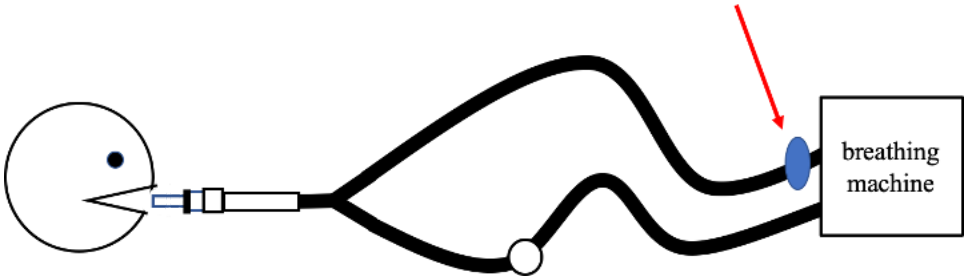
[363] This is the general idea behind the "CPR Mode" setting on the Hamilton T1

One last recap and then we'll move on. With a deteriorating patient on the vent, try to keep it simple and work through four steps: ABCs, alarms, Three Big Things, and then the extra things (pressures, trends, graphics, etc.). If after that an issue persists, consider removing the patient from the vent and bagging by hand (with a PEEP valve, if available). And if other assessment techniques such as ultrasound are available, they can also be used to further investigate causes of deterioration. In the event that the patient arrests while on the vent, cancel out patient-triggers and increase the high-pressure limit.

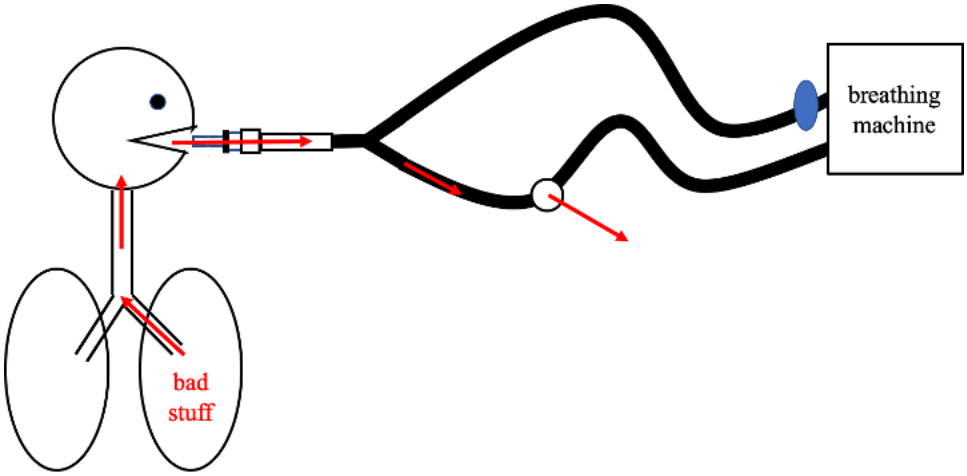
Additional Concepts Round Two

Filters[364]

Filters are used in mechanical ventilation to prevent infectious gunk from transferring from one spot to another. In the transport setting we generally use in-line filters that simply fit into the vent circuit. While there are a few possible options as to where we place the filter, it is commonly put at the connection between the machine and the vent circuit (i.e., the inhalation side of the system):



The filter placed here essentially keeps stuff at the machine from getting to the patient. Which is fine, just recognize that it doesn't keep bad stuff at the patient from getting to us and our coworkers:

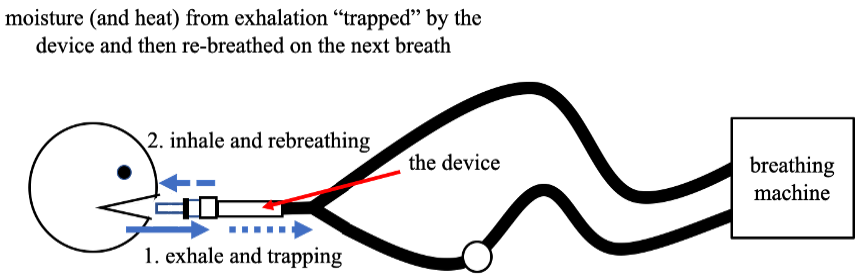


Now we could work around that by placing the filter at the patient's face/ endotracheal tube or even on the exhalation side of things, but the face option will increase mechanical dead space and the exhalation side option may not be available with our transport vent.[365] That said, placing a filter near the tube may be warranted in certain cases (tuberculosis, flu, COVID, etc.), just know that in addition to the dead space issue it can also impede the movement of air. But if we have a patient with something that we don't want to breathe in and neither of these strategies is appropriate or possible, be sure to mask up!

[364] Wilkes, 2011a & 2011b - He gives a very in-depth discussion of both filters (this section) and humidifiers (next section)
[365] The impact of adding the humidifier (and other devices) to the circuit is discussed in both **Dead Space** and the **Appendix**

Humidifiers[366] [367]

Humidification of air is important in mechanical ventilation because dry air can cause damage to the lining of the respiratory tract. No need to get into the details here, just know that absent any contraindications we ought to try and add some degree of humidification to the air we push into the patient's lungs. We typically do this in transport by placing a humidification device called a heat & moisture exchanger (HME) between the endotracheal tube and wye of the vent circuit. Placing the device further up on the inhalation side of the circuit would not work, as the device functions by trapping moisture (and also heat) from exhaled air and allowing it to be blown back into the patient's airways on the subsequent breath:



The humidifier is often the biggest contributor to mechanical dead space (as outlined in the **Appendix**), but it ought to be used unless we have good reason not to. One of these good reasons not to would be a small **Tidal Volume**, such as with kids or **Lung Injury** patients.[368] In these situations, we want to minimize mechanical dead space as much as possible. Now there are smaller devices designed for pediatrics and here's the basic idea on that: humidifiers are rated to provide humidification for a certain amount of tidal volume; higher value corresponds with more space needed within the internals of the device and, therefore, more dead space.[369] To make this clear, let's look at info from one particular product line:[370]

Gibeck® Humid-Vent® HME

ITEM CODE	DESCRIPTION	TV RANGE (ML)	MOISTURE OUTPUT (MG H2O/L)	WEIGHT (G)	RESISTANCE (CM H2O)	DEAD SPACE (ML)	CASE QUANTITY
10011	Humid-Vent Mini	15-50	30, Vt = 20 mL	4.5	0.9, 10 lpm	2.4	30
11112	Humid-Vent 1	50-600	30.5, Vt = 0.2 L	9.4	0.3, 20 lpm	10	50
11132	Humid-Vent 1 port	50-600	30.5, Vt = 0.2 L	11.6	0.3, 20 lpm	14	25
13312	Humid-Vent 2 port	150-1500	28, Vt = 0.6 L	20.9	0.8, 60 lpm	29	20
14412	Humid-Vent 2S	150-1500	28, Vt = 0.6 L	19.8	0.8, 60 lpm	29	20
17731	Humid-Vent 2S Flex–sterile	250-1500	28, Vt = 0.6 L	26.4	0.8, 60 lpm	54	20
17732	Humid-Vent 2S Flex–clean	250-1500	28, Vt = 0.6 L	26.4	0.8, 60 lpm	54	20

we see here that more capacity for humidification means more dead space

[366] Yartsev, 2021a - Navigate here for a discussion of the passive style devices used in the transport setting
[367] Gillies & friends, 2017 - This Cochrane Review has determined that passive humidifiers are comparable to actual humidification in providing therapeutic benefit and avoiding primary complications (airway obstruction, pneumonia, mortality); while they admit that more research is needed, it's good to know that these devices do have demonstrated value
[368] Hinkson & friends, 2006 - This article looks at the effect of dead space in mechanically ventilated patients, we'll visit it again in the **Appendix** and will focus in on applying the idea to treatment of pediatric patients
[369] Which means we could theoretically use a smaller-sized humidifier for an adult patient ventilated via a low-volume strategy
[370] Teleflex, 2019 (images) - Just to be clear, no relationship or conflict of interest here; it's just really nice how they lay out all the product info like this for us to talk about

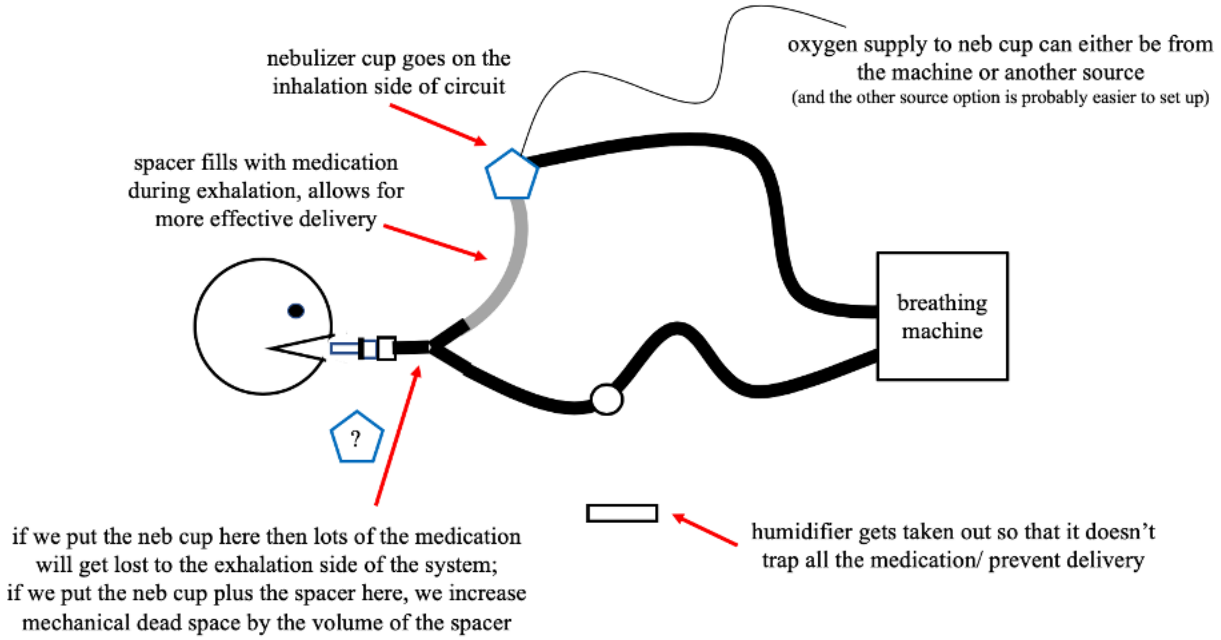
Second good reason not to use a humidifier would be the concurrent use of nebulized medications.[371] We want those drugs going into the patient, not getting absorbed by the device. While we could theoretically place the in-line nebulizer between the endotracheal tube and the humidifier, that would result in decreased medication administration unless we also added in a spacer. But then we'd have a huge amount of dead space and we already established that we want to cut down on that whenever possible. Also, the need for a humidifier is less with a nebulized medication because we are actively pushing moisture into the airways along with whatever medication is being given. It is, however, OK to remove the humidifier for administration of a nebulized drug and then reattach it as soon as that is done.

One other situation in which we ought to exercise concern with a humidifier would be increased secretions, as the device can get clogged up to the point where it impedes airflow. This isn't a case in which we never use a humidifier, rather it's one of those cases where we need to be aware of potential problems. Increases in **Peak Inspiratory Pressure** in **Volume Control** or decreases in exhaled tidal volume in **Pressure Control** would likely be our first indication of an airflow problem of this sort.[372] If this happens and we are worried about a humidifier getting clogged up, we can either remove the device or replace it with a fresh one.

Very last thing about humidifiers before moving on: while all of these devices provide some filtration of exhaled air, certain devices may even be classified as both a filter and a humidifier. This could potentially mitigate the escape of infectious material from the patient into the ambient air via the exhalation side of the vent circuit as we drew out in the last section.

In-line Nebulization

Just to demonstrate a few things about why we do nebs the way we do, let's look at some setups of how the system looks when we nebulize a medication through the vent circuit. Recognize that there is some variation between models:[373]



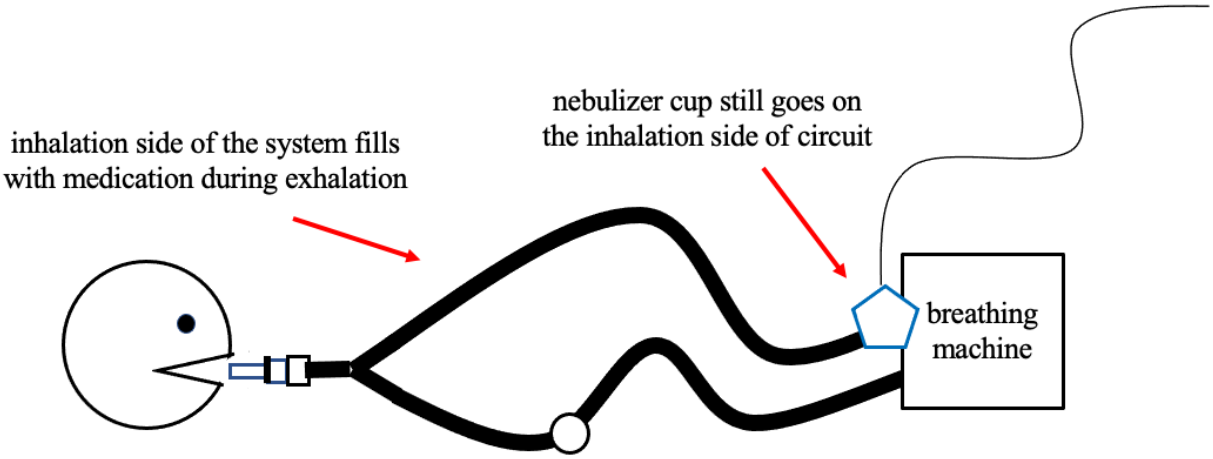
[371] And see the very next section for a discussion of **In-line Nebulization**

[372] Since we don't routinely monitor flow in the transport setting, we instead have to look at other parameters to diagnose these kinds of air movement problems

[373] Dhand, 2017 - And for more info on placement of the nebulizer and bias flow (which we don't get into here), refer back to **Triggers** or take a read of this article

That should be clear enough, but just to expand on a few points: we may need adapters and extra vent tubing to make this work, so we should plan ahead and have that stuff available in pre-built kits. The spacer is important, as medication will be lost to the exhalation side of the circuit if it isn't there.

Another setup with the nebulizer at the machine rather than embedded in the circuit might look like this:



Some machines recommend specific changes to settings to facilitate this process, read up on that or have a chat with the manufacturer's rep for details about a particular machine. And then don't forget to remove both **Humidifiers** (the HME ones) and **Filters** to allow for more effective delivery of aerosolized medications.

Prone Ventilation

Prone ventilation is when we lay our ventilated patient face down on the bed or stretcher. Arguments and evidence in favor of prone ventilation include things like better ventilation/ perfusion (V/Q) match, improved **Oxygenation**, more effective **Ventilation**, etc.[374] That said, prone ventilation isn't for everyone, studies are shrouded a bit by bias, and efficacy seems to be related to early implementation, time of application each day (16 hours per day!), and severity of hypoxemia (i.e., proning has benefit when oxygenation is a major issue).[375] When we are called to transport a pronated patient, there are some logistical limitations to the process. Much of what we do requires access to the patient's front side and many of the tools we use in medicine are designed with the supine patient in mind. All that said, it is likely that we will see more of this in years to come so it makes sense to do a quick survey as to where things are at in regard to prone ventilation in the field.

Prone ventilation has been mostly studied in patients with ARDS. Given that ARDS isn't something we commonly diagnose or come across initially on scene runs, it seems likely that most of our prone ventilation will be done in the context of interfacility transfers. Which is good, because the process of getting someone pronated with an endotracheal tube and vent in place isn't the fastest thing we could do and managing an airway on an already-pronated-patient presents its own complications. So interfacility transfers of ARDS folks seems to be where we will most likely be using this technique as critical care transport providers.

We mentioned before in our section on **Lung Injury** that recruitment of alveoli and maintenance of this recruitment is very important. While it may be tempting to simply flip a pronated patient over for transport and then let the receiving facility re-pronate them, this could potentially set progress back quite a bit, so we want to do what we can to keep our actions in line with the overall clinical course. That said, many treatment guidelines or algorithms for this sort of thing include cyclical proning on some sort of schedule. It may be worth timing

[374] Koulouras & friends, 2016; Henderson & friends, 2014 - For details on the benefits of prone ventilation, take a look at either of these articles

[375] Bloomfield & friends, 2015; Ehrmann, 2026 - Refer to either article for insight on research that has been done and recommendations for what ought to be investigated moving forward

these transfers in line with transport capabilities (i.e., with no capacity to transport a prone patient, simply wait until it's supine time and make it happen then).[376]

When it comes to the physical process of flipping someone over, there are a number of techniques and tools that run the gamut from a RotoProne bed[377] to simply using a flat sheet or slider.[378] Proning can also be performed at the time of transfer from one bed or stretcher to another. For example, let's say we are going from a hospital that doesn't do this to one that does; we could facilitate this at either end of the transfer.[379] This means that even if we don't transport a patient in a prone position in our vehicle, we may still get caught up in the process at some point.

A few considerations about transporting a pronated patient: access to the airway may be difficult or impossible, access to the anterior chest wall (for EKGs, assessment of heart and lung sounds, needle thoracostomy, etc.) will be limited, and stretcher or sled configuration may dictate that the patient be horizontal. For all of these reasons (and probably a great many others), it may be quite some time until certain programs and crews decide to attempt this, but rest assured that it has been done already and will likely become more common in years to come.[380]

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[376] Olveira & friends, 2017 - And as one example of that, take a look at this protocol for prone ventilation; it also goes into detail on how to carry out the physical maneuver and discusses many of the concerns that could potentially arise along the way

[377] Arjo, 2020 - Manufacturer's content on the RotoProne bed, just for those who are curious about it

[378] Critical Care & Major Trauma Network, 2015; Critical Care Cardiff, 2017 - Two YouTube videos that demonstrate proning a patient

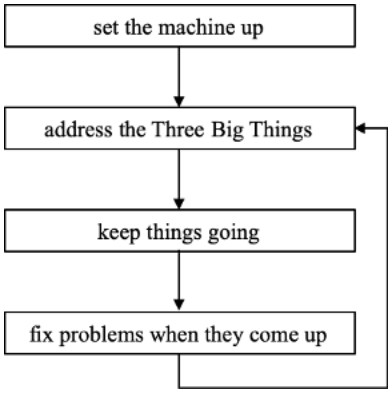
[379] Hospital Direct, 2017 - Another YouTube video that shows the maneuver while moving a patient between surfaces

[380] Boon & Boon, 2018 - Good overview of prone ventilation and its application in the transport setting; they also have a video at that same link that shows a one-person technique for flipping a patient on a stretcher

A Proposed Protocol/ Flowchart

The goal of this learning experience is that we will know enough about vents so that we can understand why we make changes and how those changes affect our patients. Working towards that end, it may help to have a template on hand while managing a patient.[381] We've tried to create an algorithm that covers all we've talked about up to now, that is generic enough to apply to different machines, and that fits on two opposing pages so that it can easily be utilized as a reference in the field.[382] It's here to help folks work towards a higher level of competency or to simply take some of the load off of one's mind when things get busy on scene or in transport.

The basic idea of the flow is something like this:



[381] Wright, 2014; Nagler & Chiefetz, 2019 - Throughout this manual we've cited a number of different resources, but here are two of them that summarize the steps of mechanical ventilation from start to finish; they vary somewhat from what we've outlined, but provide another perspective on moving through all the steps in a systematic fashion

[382] Weingart, 2010 - As another example, this document that accompanies the podcast series can also be used as a step by step guide in a similar way to one we've outlined here

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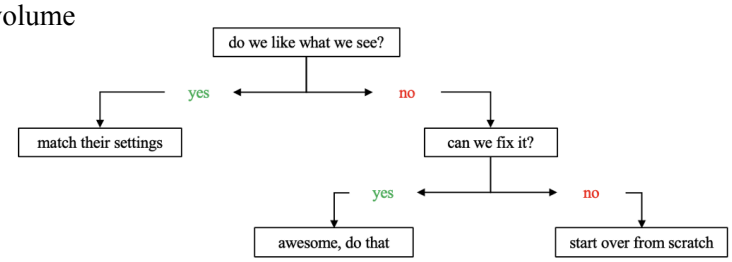
How to do Vent Stuff

I. Set the Machine Up

adult minute volume math:
IBW_{male} (kg) = 2.3(height in inches – 60) + 50
IBW_{female} (kg) = 2.3(height in inches – 60) + 45.5
MV ≈ 100ml/kg IBW/min

TV goal for all patients:
TV = 6-8ml/kg



1. prep
   a. get a report from sending
   b. do some arithmetic: IBW, tidal volume, minute volume
   c. assess the patient
   d. consider a strategy
   e. check circuit, attach $EtCO_2$ and filter; consider need for humidifier and/ or suction
2. determine settings
   a. patient already on vent (see algorithm, right)
   b. from scratch
      i. turn on machine and leave at default mode and control (unless good reason exists to do otherwise)
      ii. dial in desired tidal volume for 6ml/kg (or pressure control at 10-15cmH_2O)
      iii. adjust respiratory rate
         1. adults: to minute volume goal
         2. kids: using a reference range
      iv. adjust I-time using a reference range
      v. leave all other settings at machine defaults unless one of these considerations applies:

age description	age (years)	rate	I-time (s)
infant	0.083 (1 month) - 1	30 - 53	0.3 – 0.7
toddler	1 - 2	22 - 37	0.4 – 0.9
preschooler	3 - 5	20 - 28	0.5 – 0.9
school-aged child	6 - 7	18 - 25	0.6 – 1.1
big kid	8 - 9	17 - 25	0.6 – 1.2
preadolescent	10 - 12	14 - 23	0.7 – 1.4
adolescent	12 - 15	12 - 20	0.8 – 1.7
adult	16 and up	12 - 20	0.8 – 1.7

strategy	things to do
obstruction	decrease I;E ratio (1:≥5) by decreasing RR (and maybe I-time also), then titrate TV (or pressure control) up to maintain VE as able; consider less PEEP
hypotension	limit PEEP; increase TV and then decrease rate to maintain VE
acidosis	use high end of tidal volume (8ml/kg IBW); increase RR: pre-intubation rate, to get prior/ goal $EtCO_2$, or double normal value
lung injury	higher PEEP

3. initiate ventilation (clamp tube if concerned with de-recruitment)

II. Address the Three Big Things

parameter	assessment	normal	actions
oxygenation	SpO_2	93-99%	*low*: consider position & suction, increase FiO_2, increase PEEP, increase I-time, consider pathophysiology/ medications, think V/Q mismatch *high*: decrease FiO_2 unless contraindicated (i.e., anemia, shock, etc.)
ventilation	$EtCO_2$	35-45mmHg (low end for TBI)	*any abnormal value*: consider etiology &/ or patient compensation for acid-base imbalance (may be appropriate) *low*: consider perfusion status; decrease RR, then decrease TV; consider dyssynchrony *high*: increase TV (monitor Pplat), then increase RR; consider permissive hypercapnia
	MV	≈ 100ml/kg IBW/min (2x with acidosis)	*low*: increase TV (monitor Pplat), then increase RR; consider permissive hypercapnia *high*: decrease RR, then decrease TV; consider dyssynchrony
comfort	RASS, ANVPS	no dyspnea & no desynchrony	*extreme end*: analgesia & sedation *fine tuning*: consider dyssynchrony, address triggers, lengthen I-time (I:E ratio closer to 1:1), trial different mode/ control

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III. Keep Things Going

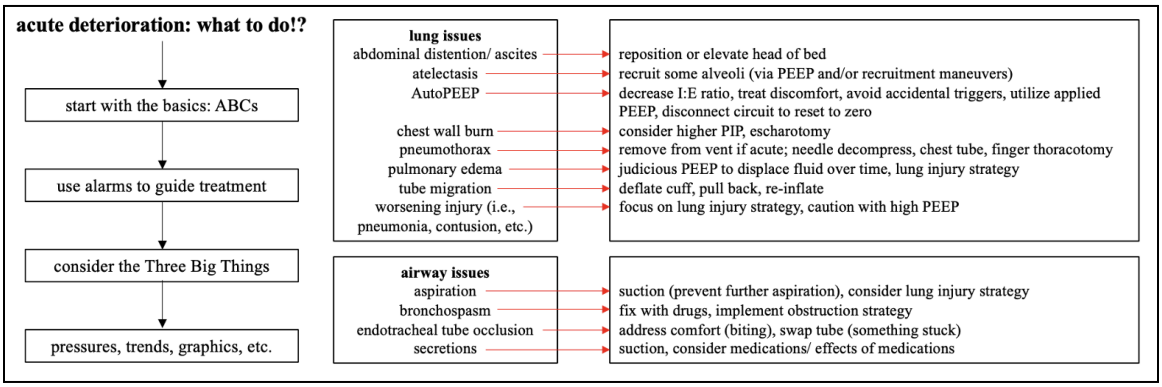
1. set (and troubleshoot) all alarms
2. consider pressures (every time vitals get reassessed)

parameter	normal	action(s)
peak inspiratory pressure	<35cmH_2O	consider potential causes (**lung** and **airway issues**), check Pplat decrease TV (or PC parameter)
plateau pressure	<30cmH_2O	consider potential causes (**lung issues**) decrease TV (or PC parameter)
AutoPEEP	none	decrease I:E ratio/ extend E-time (lower RR, shorter I-time) consider triggering, trial VC if in PC, adjust PEEP disconnect vent to allow exhalation
driving pressure	<15cmH_2O	decrease TV or pressure control consider more PEEP and permissive hypercapnia consider recruitment maneuvers
mean airway pressure	not applicable	monitor for trends and investigate further

3. make adjustments moving forward

strategy	things to do
general stuff	if oxygenation is all good, go down on FiO_2 (maybe all the way to 0.40) and reevaluate consider increasing TV, but maintain safe Pplat and acceptable driving pressure
obstruction	use drugs (in-line neb treatment(s), consider Ketamine for analgesia/ sedation, etc.) ensure no AutoPEEP develops if hypercapnia develops and/ or no AutoPEEP noted, consider moving towards normal I:E ratio
hypotension	use caution with PEEP to improve oxygenation (can decrease preload) consider fluids and/ or pressors (i.e., resuscitate) if perfusion improves, consider working towards normal settings to avoid higher airway pressures
acidosis	maintain increased VE goal (200ml/kg IBW/min may be necessary) also consider Winter's Formula and/ or $EtCO_2$/ PCO_2 gap to guide treatment
lung injury	consider titrating TV down to 5ml/kg, then 4ml/kg to maintain driving pressure <15cmH_2O increase PEEP to maximize oxygenation, consider stepwise approach consider recruitment maneuver(s) if hypoxia persists also consider allowing permissive hypercapnia

IV. Fix Problems When They Come Up



More on Pediatric Ventilation

In general, the guidance previously discussed covers how to ventilate pediatric patients of all ages, but it's worth digging in on some specifics and addressing a few concepts that frequently come up with pediatrics. Consider this section an addendum once the basics have been mastered.

Minute Volume for Pediatrics

The most notable difference between how we ventilate children and how we go about it with adults is the strategy for **Minute Volume**. In adults, we tend to quantify the thing; but with pediatrics, it's a bit less rigid. This is noted in **A Proposed Protocol/ Flowchart**, but we'll get into the details here.

For the adult patient we advocate using a minute volume goal of 100ml/kg IBW/min at baseline, but for pediatrics we state to utilize a reference card.[383] This means we use a **Tidal Volume** based on actual or estimated weight and a **Respiratory Rate** from some pre-made reference and therefore end up with a minute volume that is dependent on these chosen values. There are, however, strategies with minute volume goals by age range as so:[384]

Age-Based Minute Volume Goals	
age group	***ml/kg/min***
adult	100
child	150
infant	200
newborn	300

These values originate from the idea that pediatric patients have a higher rate of metabolism than adults and that the difference, compared to adult values, increases as age approaches zero. To say it another way, newborns and infants have more carbon dioxide to blow off than adults, therefore their need for ventilation is higher (and again, this is all in terms of size or weight or body surface area). This concept is encouraged by the observation that respiratory rates also increase as age approaches zero (i.e., babies breathe faster than adults). Another factor is that more total volume (i.e., minute volume) is needed as the ratio of **Dead Space** to tidal volume increases; and that ratio increases as age gets closer to birth (i.e., for younger patients).[385] For all of these reasons, the weight-based minute volume goal increases the younger the patient gets.

We recommend initiating mechanical ventilation in a pediatric patient using a tidal volume based on estimated or actual weight and a respiratory rate in line with norms for the patient's age. Moving forward, however, we can still consider these weight-based goals for minute volume during reassessment. For example, if we are ventilating a six-year-old child with an estimated weight of 24kg and notice that our exhaled minute volume is 2.4L or 2400ml or 100ml/kg IBW/min, we may want to reconsider (i.e., we are likely below goal). Ideally we'd simultaneously evaluate end-tidal carbon dioxide, but that may not always be available and therefore knowing these approximate ranges for minute volume can serve as an additional check.

Also note that there are cases in which a rate above the normal reference range for a given age is expected. In these cases, consideration should be given to the idea that there may be a need for additional volume/ a higher rate/ an increased minute volume above a normal goal. The classic example that we've

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[383] This was discussed initially in **Respiratory Rate**, mentioned again in **A General Vent Strategy**, and then is explained in detail towards the end in the **Appendix**

[384] Gardon & friends, 2023 - The PRoVENT-PED study determined these ranges (which we have simplified slightly); there are also comparable suggestions in Vent Hero by Charles Swearingen, but he cites higher ranges per age group

[385] This is explained in detail with some math in **Mechanical Dead Space**

already discussed is a state of **Acidosis**, but other causes might include congenital conditions/ abnormalities, inborn errors of metabolism, infection, ingestion/ intoxication, etc. As with adults, if we are unsure it is generally preferable to overventilate than to underventilate.

As for how to ascertain a weight for a pediatric patient (because we still do want to use a weight-based tidal volume) there are a few options: actual weight, estimated weight by formula, and estimated weight by length-based resuscitation tape. We discussed this back in the section on **Ideal Body Weight** and identified a few formulas for different age ranges. Just as with adults, the idea holds that a pediatric patient of a certain height or length likely has lungs of comparable size to another pediatric patient of equal height or length, even if actual weight between the two differs significantly. For that reason actual body weight may not be the best parameter from which to determine vent settings with extremes of size in either direction.

Pressure vs. Volume

In transport medicine we often teach to use **Volume Control** with adults and **Pressure Control** with pediatrics, but this simplification warrants a bit of discussion. We said this once already, but for nearly all patients any combination of mode and control can be used effectively, so long as we understand what exactly it is that the vent is doing and what the values it gives us actually mean.[386] This is to say that it's completely OK to ventilate a kid with volume control ventilation. And in fact, that's often the case outside of the transport setting.

The argument in favor of pressure for pediatrics is two-fold. First is that it helps us to avoid barotrauma and/ or volutrauma by giving too much air. Second is that it helps us to avoid hypoventilation if we fail to consider the effect of **Dead Space**. Both of these things can be easily mitigated. On the flip side of the argument, there is some thought that pressure control ventilation in pediatrics (and particularly in infants and neonates, which we discuss next) yields worse outcomes in certain situations than volume control.[387] While the argument could most definitely be made that this likely reflects utilization and/ or strategies and not the method of control itself, the point is that pressure control should not be assumed to be superior to volume control.

Regardless of which method of control is utilized, we always want to have a weight-based tidal volume calculated out so that we can compare exhaled tidal volume to this goal and adjust as needed. A target of 6-8ml/kg estimated weight is still an appropriate starting point for pediatric patients of all age ranges. Just keep in mind, however, the following concepts: exhaled tidal volume is the preferred data point for evaluating how effectively we are achieving that tidal volume goal, the accuracy of exhaled tidal volume measurements decreases with volume (i.e., is less accurate with smaller volumes), and there is stretch in the vent circuit that may result in overestimation of delivered volume with volume control ventilation.[388]

And then the last point to make regarding pressure and volume in pediatric ventilation is that narrow airways and smaller endotracheal tubes may result in observed or measured parameters that are out of range.[389] For example, volume control ventilation via a small-diameter endotracheal tube may result in a high **Peak Inspiratory Pressure** due to increased resistance to airflow.[390] Likewise a pediatric patient ventilated in pressure control may need a higher **Driving Pressure** to achieve a goal exhaled tidal volume. This may be further complicated by the fact that we sometimes choose to intentionally limit peak pressures, such as with neonates or in pediatrics with severe lung injury, although we should recognize that the data here is limited.[391] As always, recognize that **Plateau Pressure** is the better measure of alveolar safety.

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[386] Kneyber & friends, 2017 - The group of experts that make recommendations on the specifics of mechanical ventilation for pediatrics share this sentiment

[387] Sarkar & friends, 2023 - This paper concludes that volume may be best in pediatric ventilation when oxygenation is of concern, but this is at the expense of higher airway pressures

[388] This was discussed already in **Dead Space** and quantified in the **Appendix**

[389] Woodward, 2025 - More on Poiseuille's Law and how it applies to airway resistance

[390] Resistance was discussed previously in **Compliance & Resistance**

[391] Kneyber & friends, 2017; Chakkarapani & friends, 2020b - Each touches briefly on the idea of peak pressures in pediatric mechanical ventilation and cases in which limiting levels may be indicated

Infants & Neonates

When we discuss mechanical ventilation in our smallest of patients, there are a few things to consider that make management and the choice of initial settings a bit different. First is that **Tidal Volume**, **Respiratory Rate** and **Minute Volume** are all highly variable:[392]

APPENDIX 4

Normal Lung Function Data for Term Newborns during the Neonatal Period

Measurement	No. of Infants Studied	Mean	Standard Deviation	Range	
Tidal volume (mL/kg)	266	4.8	1.0	2.9-7.9	
Respiratory rate (bpm)	266	50.9	13.1	25-104	
Minute volume (mL/kg/min)	266	232	61.4	0.9-3.7	**(assuming this to be 90-370ml)**

FRCpleth, functional residual capacity by body plethysmography. (Data from Milner et al. Effects of smoking in pregnancy on neonatal lung function. *Arch Dis Child.* 1999;80:F8–F14.) The tidal volume and volume were measured using a type 00 Fleisch Pneumotachograph and intrathoracic pressure with a 4-cm esophageal balloon. Babies were supine in quiet sleep. Dead space was eliminated by a bias flow of air.)

(attempted to verify assumptions via primary source, but seems to be cited incorrectly)

Also note that average tidal volume is lower than what we would typically use for adults (4.8ml/kg vs. 6-8ml/kg).

Next is that end-tidal carbon dioxide may not be as reliable of a measure to evaluate ventilatory status (compared to adults), because the arterial partial pressure of carbon dioxide (which correlates with the end-tidal reading) is more variable:[393]

APPENDIX 8

Arterial Blood Gas Values in Normal Full-Term Infants*

		Umbilical Vein (cord)	Umbilical Artery (cord)	5-10 min	20 min	30 min	60 min	5 h	24 h	2 days	3 days	4 days	5 days	6 days	7 days
pH	$\bar{X}$	7.320	7.242	7.207	7.263	7.297	7.332	7.339	7.369	7.365	7.364	7.370	7.371	7.369	7.371
	SD	0.055	0.059	0.051	0.040	0.044	0.031	0.028	0.032	0.028	0.027	0.027	0.031	0.023	0.026
P_{CO_2} (mm Hg)	$\bar{X}$	37.8	49.1	46.1	40.1	37.7	36.1	35.2	33.4	33.1	33.1	34.3	34.8	34.8	35.9
	SD	5.6	5.8	7.0	6.0	5.7	4.2	3.6	3.1	3.3	3.4	3.8	3.5	3.6	3.1
P_{O_2} (mm Hg)	$\bar{X}$	27.4	15.9	49.6	50.7	54.1	63.3	73.7	72.7	73.8	75.6	73.3	72.1	69.8	73.1
	SD	5.7	3.8	9.9	11.3	11.5	11.3	12.0	9.5	7.7	11.5	9.3	10.5	9.5	9.7
Standard bicarbonate	$\bar{X}$	20.0	18.7	16.7	17.5	18.2	19.2	19.4	20.2	19.8	19.7	20.4	20.6	20.6	21.8
(mEq/L)	SD	1.4	1.8	1.6	1.3	1.5	1.2	1.2	1.3	1.4	1.4	1.7	1.7	1.9	1.3

(Data from Bancalari E. Pulmonary function testing and other diagnostic laboratory procedures. *J Peds.* 1987;110(3):448-456; Thibeault DW, Gregory GA, eds. *Neonatal Pulmonary Care.* Reading, Mass., Addison–Wesley Publishing Co., 1979:123, Table 7-4.)
*Values from Koch and Wendel. Blood obtained through umbilical artery line. Po and P_{CO_2}, measured with Clark and Severinghaus electrodes.
SD, standard deviation; $\bar{X}$, sample mean.

Arterial Blood Gas Values in Normal Premature Infants*

		3-5 h	6-12 h	13-24 h	25-48 h	3-4 days	5-10 days	11-40 days
pH	$\bar{X}$	7.329	7.425	7.464	7.434	7.425	7.378	7.425
	SD	0.038	0.072	0.064	0.054	0.044	0.043	0.033
P_{CO_2} (mm Hg)	$\bar{X}$	47.3	28.2	27.2	31.3	31.7	36.4	32.9
	SD	8.5	6.9	8.4	6.7	6.7	4.2	4.0
P_{O_2} (mm Hg)	$\bar{X}$	59.5	69.7	67.0	72.5	77.8	80.3	77.8
	SD	7.7	11.8	15.2	20.9	16.4	12.0	9.6
Base excess (mEq/L)	$\bar{X}$	−3.7	−4.7	−3.0	−2.3	−2.9	−3.5	−2.1
	SD	1.5	3.1	3.3	3.0	2.3	2.3	2.2

(Data from Bancalari E. Pulmonary function testing and other diagnostic laboratory procedures. In: Thibeault DW, Gregory GA, eds. *Neonatal Pulmonary Care.* Reading, Mass., Addison–Wesley Publishing Co., 1979, 123, Table 7-5.)
*Values from Orzalesi et al. Mean birthweight, 1.76 kg; gestational age, 34.5 wk. Blood obtained from radial, temporal, or umbilical artery. P_{O_2} measured with Clark electrode, and P_{CO_2} calculated with use of the Siggaard-Andersen nomogram.
SD, standard deviation; $\bar{X}$, sample mean.

All of this means that our strategies for both determining initial vent settings and evaluating ventilation need to be adjusted for the infant and neonate populations. This is not to say that end-tidal carbon dioxide

[392] Goldsmith & friends, 2017 - Partial chart and the next one borrowed from this excellent textbook on mechanical ventilation of the neonate; also there is a newer vision or two of this book that is not freely available
[393] Wong & friends, 2021 - More on this variation in CO_2 with neonates in this more recent article

monitoring has no place in managing the infant or neonate that is being mechanically ventilated, rather that its ability to accurately describe the efficacy of ventilation may not be as effective as with older or larger patients.

The recommendation, therefore, is to utilize a **Tidal Volume** based on weight (estimated, calculated or actual, as discussed above) and a vent strategy that allows the patient to dictate ventilatory rate based on intrinsic metabolic need. Assessment of ventilation in the smallest of patients should consider the following:

Assessment of Ventilation in the Infant & Neonate		
	tidal volume	**respiratory rate**
below goal	no chest wall movement/ chest rise -and/ or- exhaled tidal volume below calculated goal (4-8ml/kg)	below normal or expected range, adjusted based on pathophysiology
at goal	chest wall movement/ chest rise -and- exhaled tidal volume at calculated goal (4-8ml/kg) -and- normal peak inspiratory pressure	within normal or expected range, adjusted based on pathophysiology
above goal	high peak inspiratory pressure (in the absence of a good reason, such an undersized ETT &/ or very short I-time) -and/ or- - exhaled tidal volume approaching or > 8ml/kg	above normal or expected range, adjusted based on pathophysiology

And in light of this chart, recall that **Ventilation** is the product of **Tidal Volume** and **Respiratory Rate**. While we evaluate each of these components independently, know that we can adjust either one to impact **Ventilation**.

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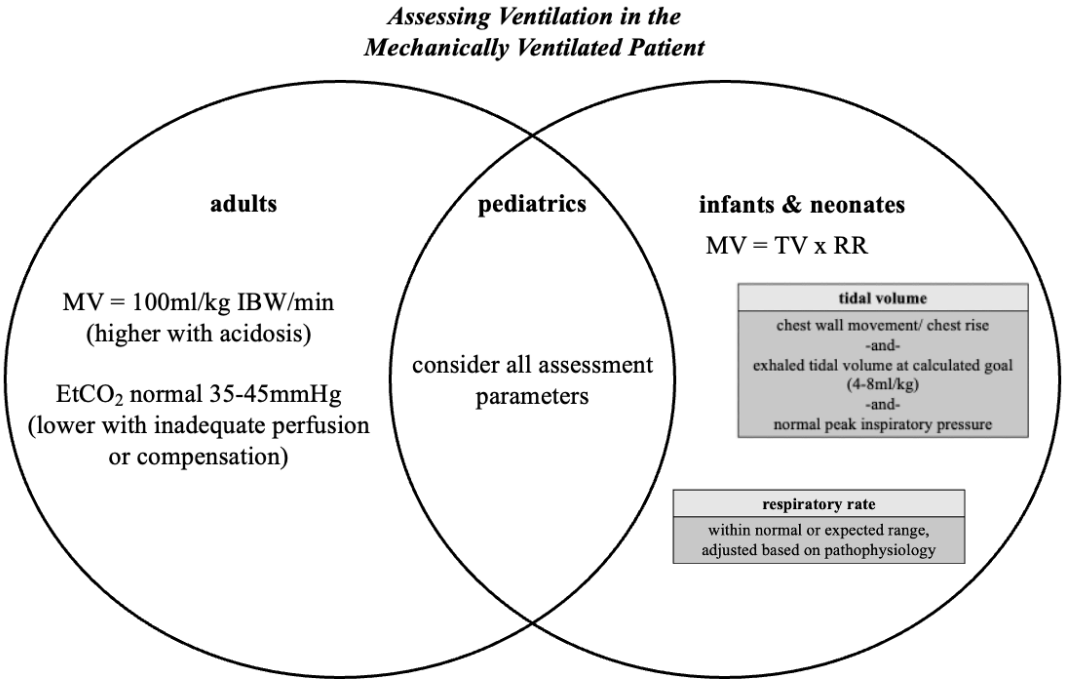
Pediatric-to-Adult Spectrum

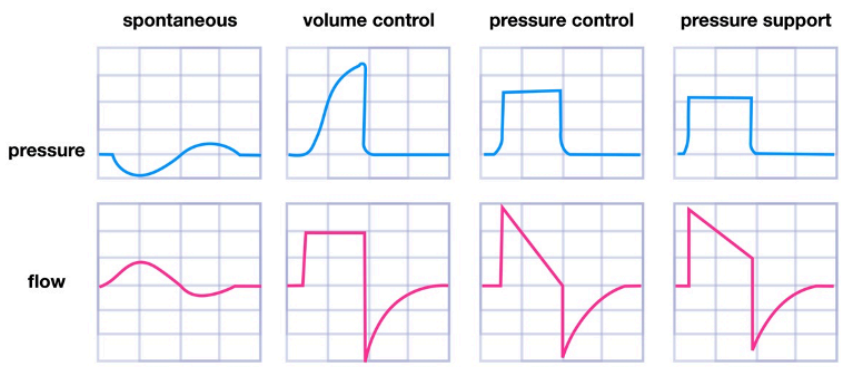
The very last point to make in reference to mechanical ventilation of the pediatric patient is that the stark contrast in recommendations for management of the adult versus infant/ neonate populations is less obvious in the middle ground. For example, while end-tidal carbon dioxide monitoring is often considered standard of care with adults and may not be as reliable with infants/ neonates (see discussion above), it may have a place with toddlers or school-aged children (or any other age range for that matter). Same goes for utilizing a calculated **Minute Volume** goal based on **Ideal Body Weight** - the concept seems appropriate in the adult population, but less so in the infant/ neonate group. That said, it can be a helpful concept for pediatric patients of all ages and can still be applied to older pediatrics who are approaching the size and characteristics of an adult. All that said, the primary difference here is that with adult patients we often assess ventilation by way of end-tidal carbon dioxide and a weight-based minute volume goal and with infants/ neonates we assess ventilation by directly assessing the two contributing components - **Tidal Volume** and **Respiratory Rate**. Consider combining both strategies for pediatric patients in general.



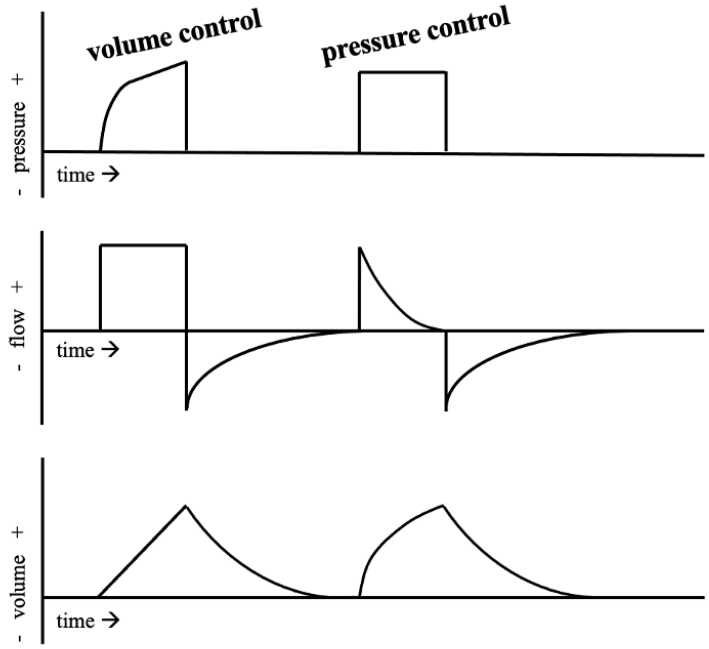
Vent Graphics

Ventilator graphics include both waveforms and loops. We'll mostly focus on waveforms here, but will provide a cursory overview of both and, as always, links for further reading on the subject. To start with, recognize that not all transport ventilators show graphics and they aren't necessarily needed to manage patients on ventilators. In fact, until very recently, most transport folks had very limited access to graphics. That said, they can provide some useful information and can help to visualize what is happening with our patients. It's also worth pointing out that nearly all modern hospital ventilators do have graphics, so even if we don't have access to them during transport, we can interpret information from those visuals at the time of transfer.

Ventilator graphics basically show how two variables interact when plotted on X and Y axes of a chart. We've already seen examples of these before, such as this depiction of pressure and flow waveforms:[394]

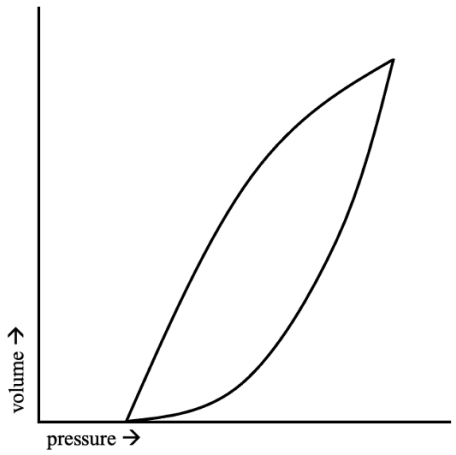


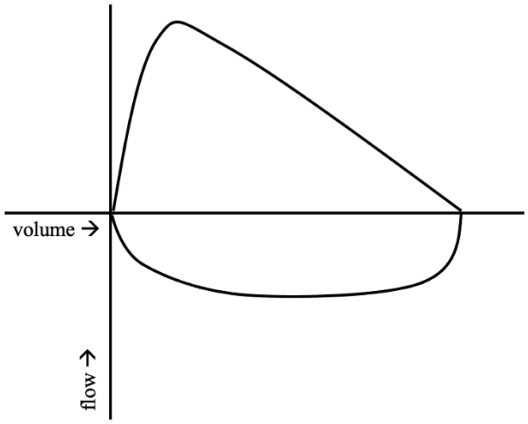
Waveforms, one type of graphic and as shown above, depict pressure, flow, and/ or volume, each graphed out over time (i.e., with time on the x-axis). Here is what some normal waveforms might look like if the vent is doing all the work and the patient isn't making a perceptible effort:



[394] This was first shown in Types of Breaths

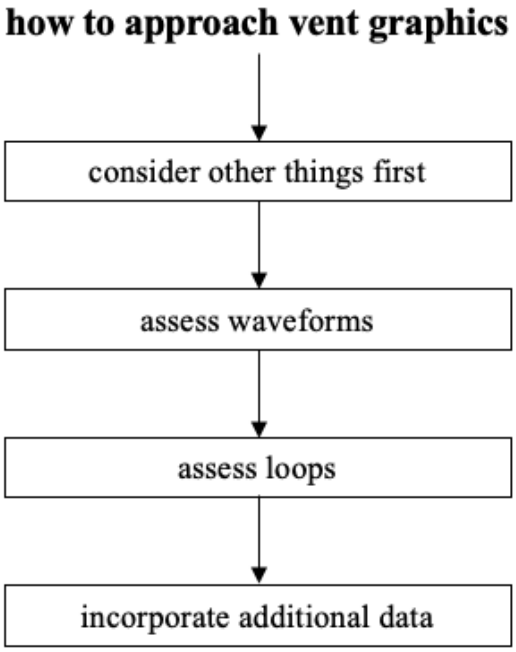
On the other hand, loops depict interactions between these three variables (pressure, flow, volume) in reference to another. For example, we may choose to look at a pressure-volume loop or a flow-volume one. Here's what the normal ones might look like, again assuming no patient effort:





These graphics, both waveforms and loops, can be used to better understand how the ventilator and patient are interacting in the context of the settings we have input. And then we can use that information to make changes in those settings to better manage the patient. Just know that this gets complicated and it takes some time to learn.

And as a bit of a preview, here is the workflow we'll work towards for how to interpret vent graphics:



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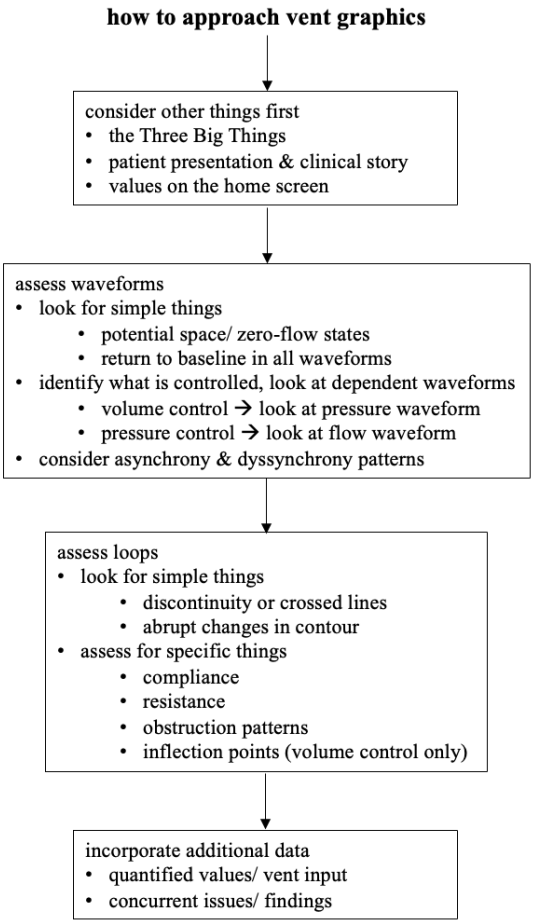
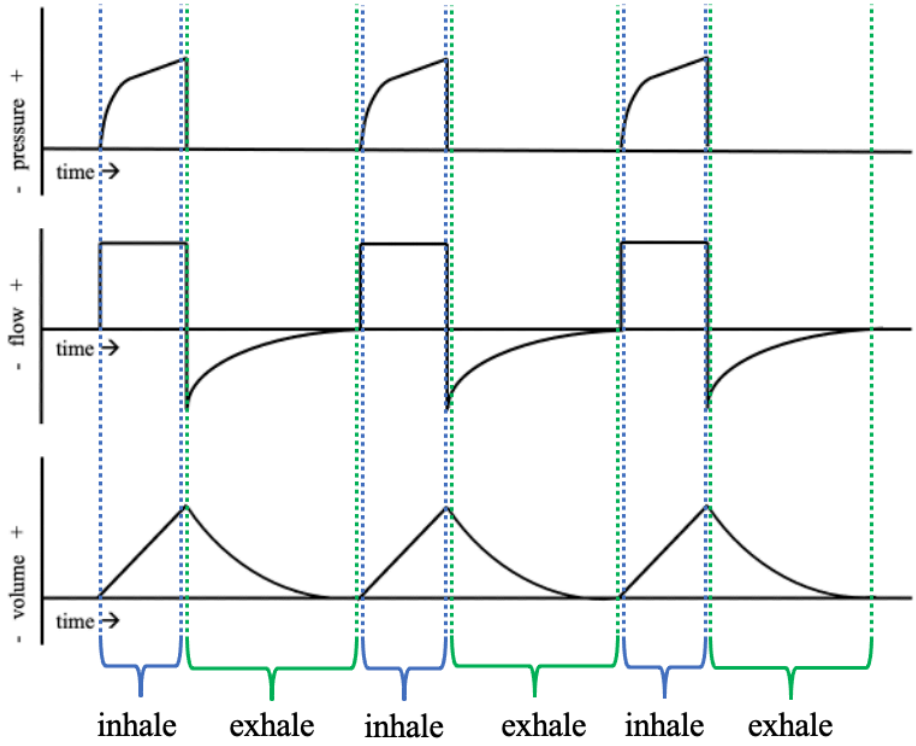
And then in more detail:

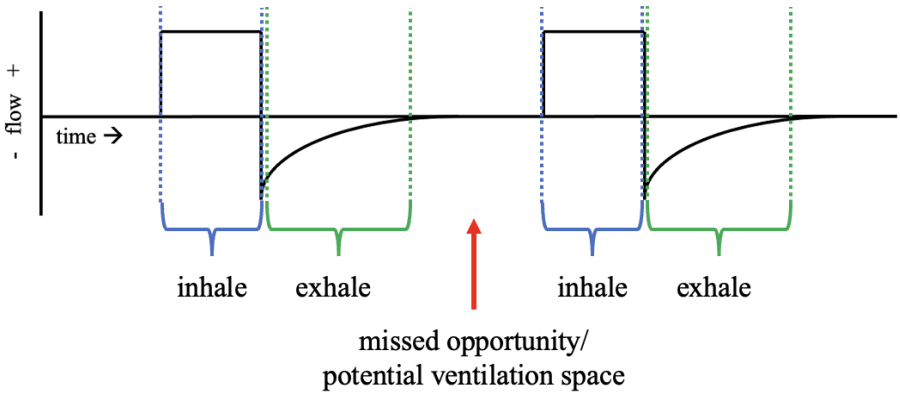


Waveforms

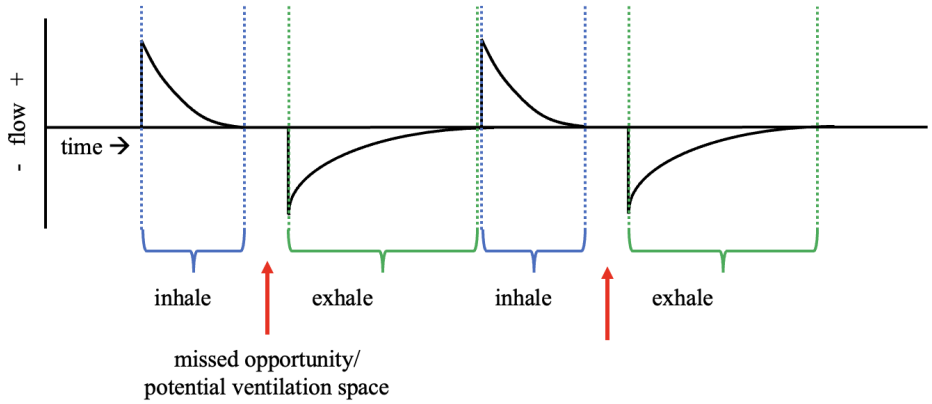
The clinical utility of vent waveforms begins with understanding the normal or baseline waveforms. Let's look at some normal waveforms and annotate a few key points. First thing to recognize is which portions of each set represent inhale and which depict exhale. And also that normal is each inhale followed immediately by an exhale. We'll show a series of waveforms in volume control here just as an example:



Presence of a gap or space between the two indicates a potential opportunity for **Ventilation** to occur. We could capitalize on this by shortening the expiratory time so that the next breath can initiate sooner. For this example, we'll show just the flow-time waveform:

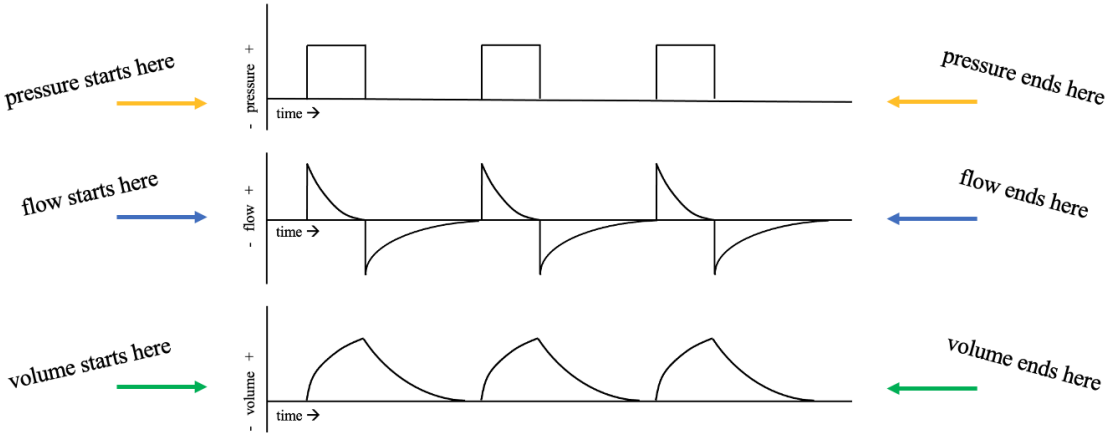


Similarly, if we look at the flow-time waveform for **Pressure Control** breaths, a gap between inhale and exhale means that we have a potential opportunity - we could optimize by shortening inhale or decreasing **Inspiratory Time** while maintaining the same **Tidal Volume** (and therefore preserve or even increase **Minute Volume**). To say this another way: if **Flow** gets back to zero before the inspiratory time runs out, there is a potential opportunity:

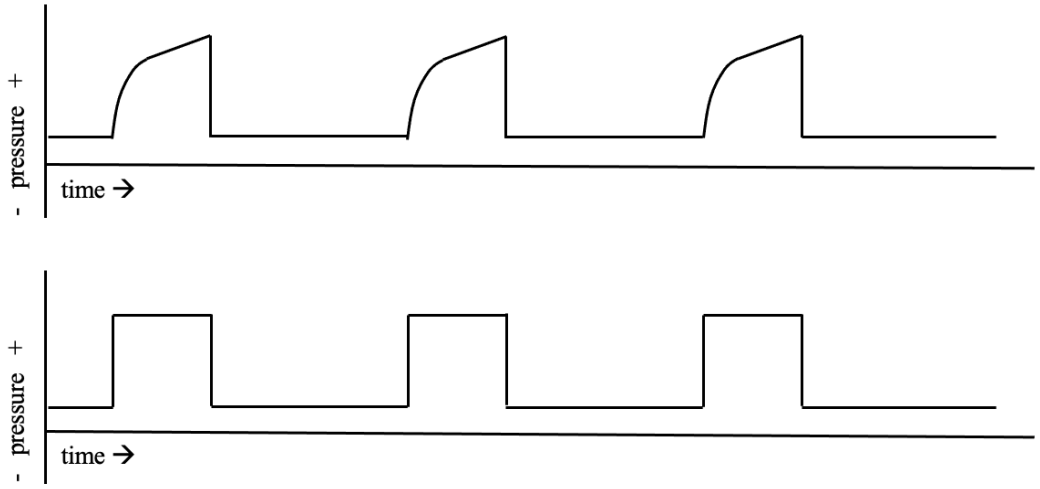


This potential opportunity matters if there is a need to increase minute volume. If the space is there, we can simply increase **Respiratory Rate** and then decrease I-time or E-time, depending on where the opportunity is seen. If no extra space is seen, on the other hand, increasing minute volume may need to occur via a higher tidal volume (or by increasing pressure control) instead.

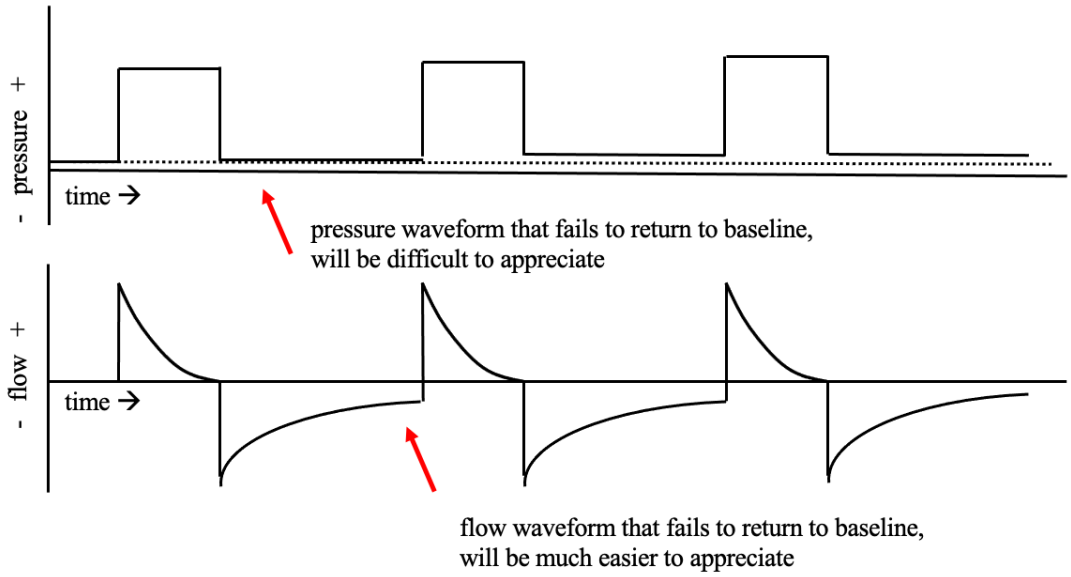
Another simple thing to check at first glance is that all the waveforms end up back at where they started. Let's look at a sequence of pressure control breaths this time:



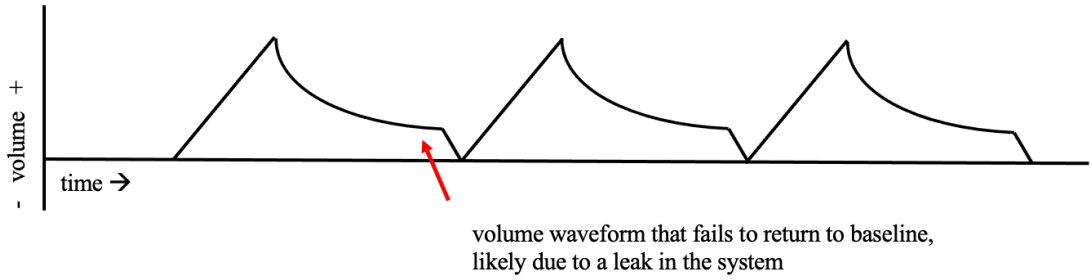
And note that for the pressure waveform, this isn't always zero. It will be whatever we have set for **Positive End-Expiratory Pressure** This is how the normal pressure waveforms look in both volume control and in pressure control with positive end-expiratory pressure added into the system:



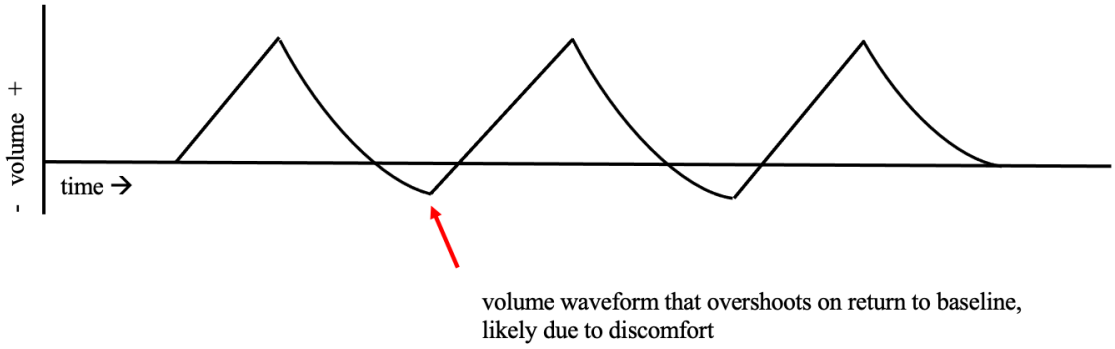
We can identify issues based on which waveform fails to return to baseline and when. A pressure waveform that fails to return to baseline would theoretically indicate **AutoPEEP** or inadequate exhalation, but this is often difficult to see on the vent's screen and would only show if it is actively increasing from breath to breath. That said, incomplete exhale or AutoPEEP is much easier to appreciate on the flow waveform and can be seen with each breath, even if AutoPEEP is not actively increasing:



And then for the volume waveform, failure to return to baseline often indicates a leak in the system, but can sometimes indicate AutoPEEP also:[395]



We might also see an overshoot in the volume waveform if there is patient effort on exhalation. This may indicate patient discomfort or prompt us to look at the other waveforms to identify concerning patterns:[396]



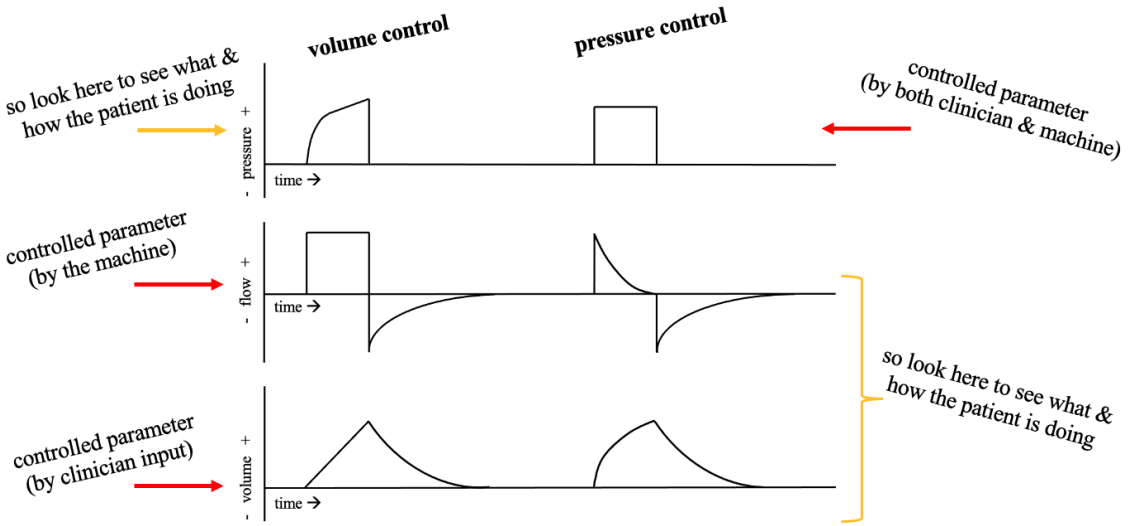
So the starting point, then, is to make sure in a quick glance that there is no prolonged time at a flow of zero and that each waveform returns back to where it started with each breath. Time spent at zero flow is wasted space for ventilation and any deviation in a waveform from a normal return to baseline represents an issue that we can fix relatively easily.[397]

[395] Mireles-Cabodevila & friends, 2021 - There are actually two other possible causes: a flow sensor error or an active exhale (by the patient) during inspiration (as breath is given by the machine) since some machines do not account for exhaled volume during inspiration

[396] More on this shortly in **Asynchrony & Dyssynchrony**

[397] We can do the same with **Loops** also - recognize normal and then quickly fix a few things that are represented by obvious deviations from that ideal pattern; that said, we've deferred a discussion of loops for now because most of the issues we can identify in loops will also be apparent in waveforms and also because we don't routinely monitor loops (this applies both in transport and in the hospital)

Next step in the analysis of vent waveforms is to figure out which waveforms we expect to be fixed or static or controlled (all terms to describe the same thing) in each mode and which waveforms might vary with patient input or interference. In general, when volume is controlled the machine also fixes flow,[398] so we then look towards the pressure waveform to see what's happening. In pressure control (which includes pressure-regulated volume control), we look at both the flow and volume waveforms to see what is happening (because pressure is fixed):[399]



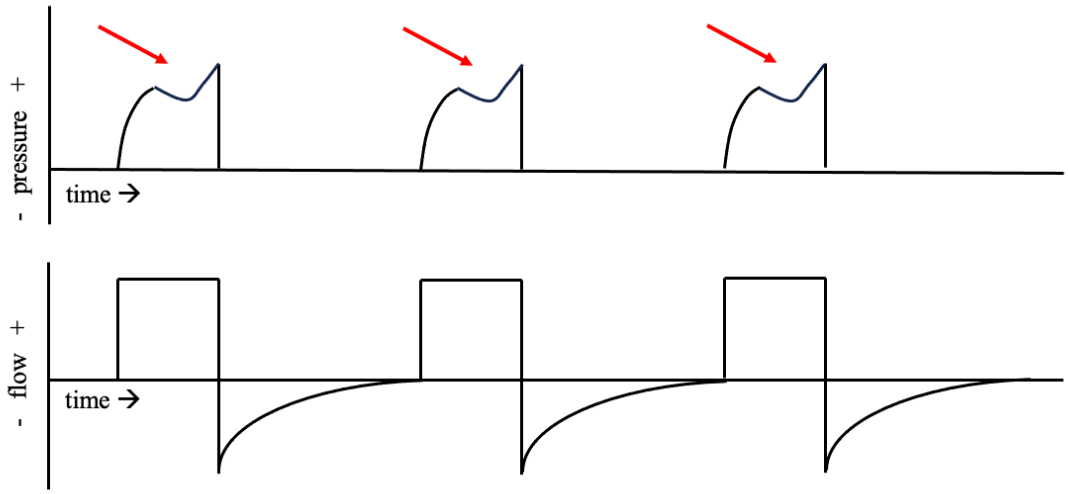
For example, let's consider the issue of flow hunger and see how it might look in both volume control and in pressure control.[400] Flow hunger is a situation in which inspiratory flow provided by the machine is less than what the patient desires. In volume control it will look like this:[401]

[398] This is what we would refer to as a square-waveform flow pattern; it is the case with most transport ventilators (i.e., most transport vents deliver **Volume Control** breaths via a square-waveform flow pattern), but is not always so in the hospital setting and may not be so with future transport vents as new things come onto the market; more on this in **Types of Breaths** and **Flow**; also note that it is only the inspiratory flow that is controlled or set, the expiratory side of things will happen based on lung dynamics and physics

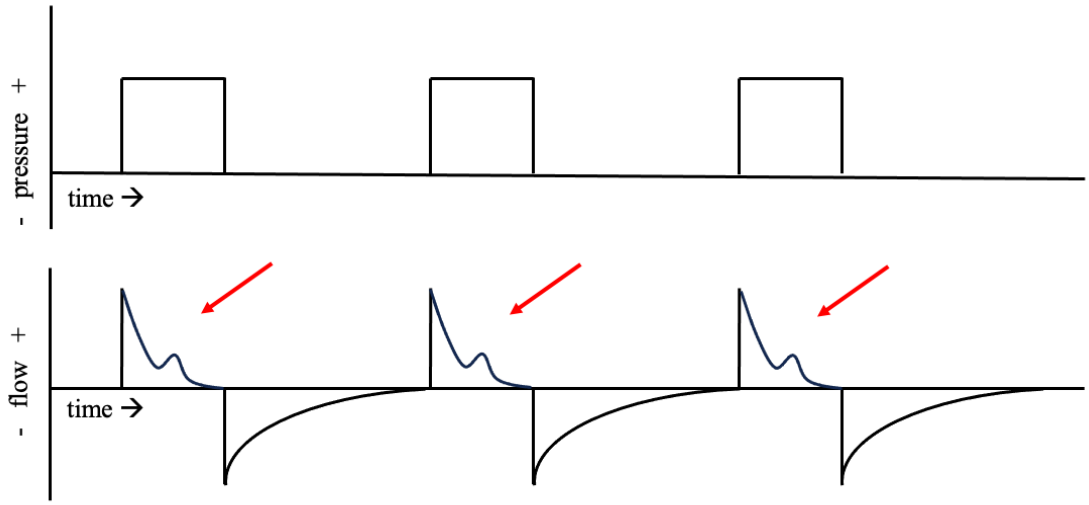
[399] Chatburn, 2026 - This idea of where to look for specific changes depending on **Control of Ventilation** is also discussed in this overview

[400] Flow hunger is often (and will be) discussed with **Asynchrony & Dyssynchrony**, but we've included a discussion here to demonstrate the idea that issues manifest in different waveforms depending on **Control of Ventilation**

[401] Emrath, 2021 - This overview of vent waveforms is from a pediatric journal, but all the details apply to adults as well

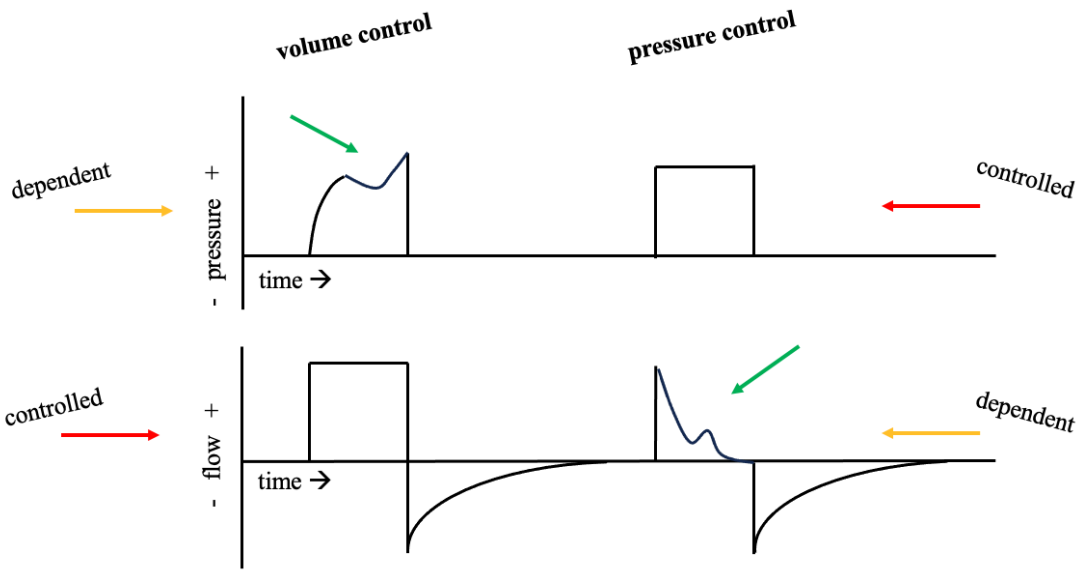


And in pressure control, it looks like this:[402]



[402] Lewis, 2020 - This video explains how flow hunger looks depending on **Control of Ventilation**

Side by side, we have this:



Another example to demonstrate where we look to see what is happening depending on **Control of Ventilation** might be when we have a decrease in compliance.[403] In volume control, we would see an increase in **Peak Inspiratory Pressure**; in pressure control we would see a decrease in **Tidal Volume**. While we don't necessarily need the waveforms for this one (as all the variables are quantifiable), it is worth appreciating how it all gets represented in the waveforms.

[403] See **Compliance & Resistance** for more

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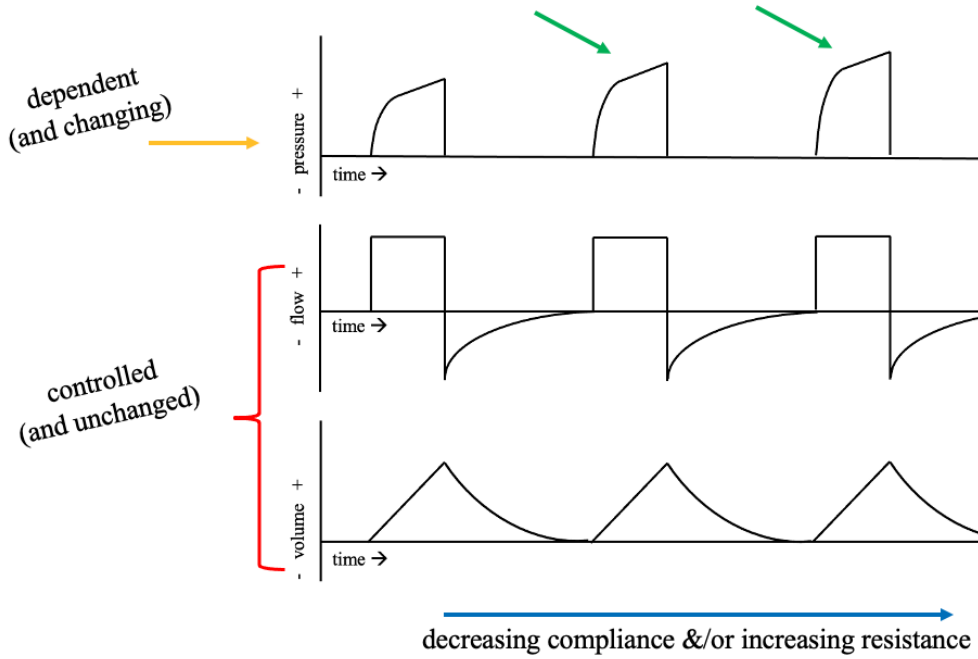
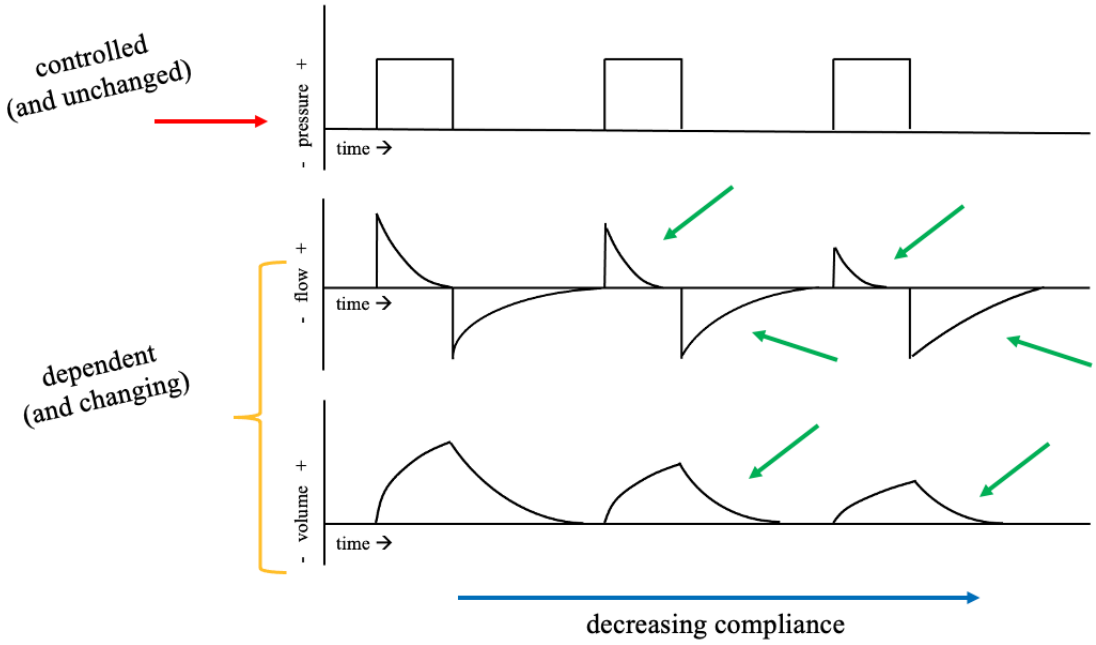
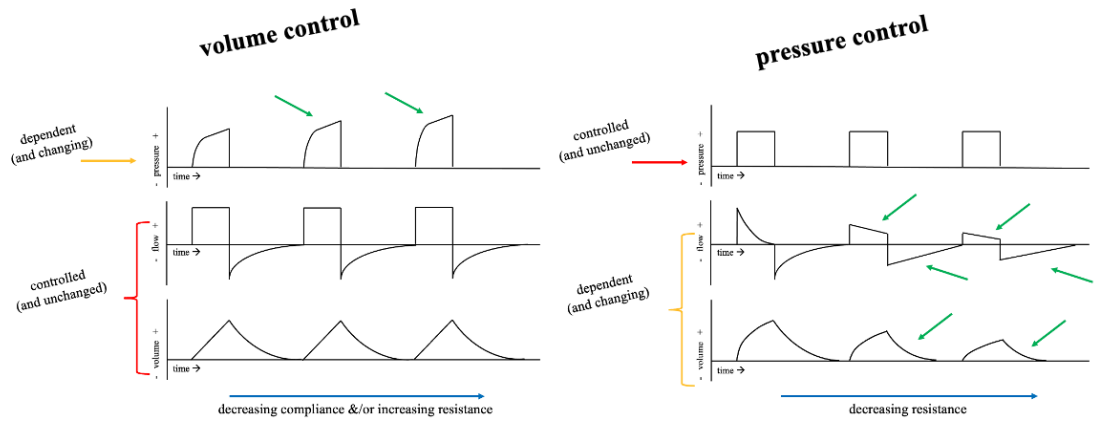
First we'll show volume control:



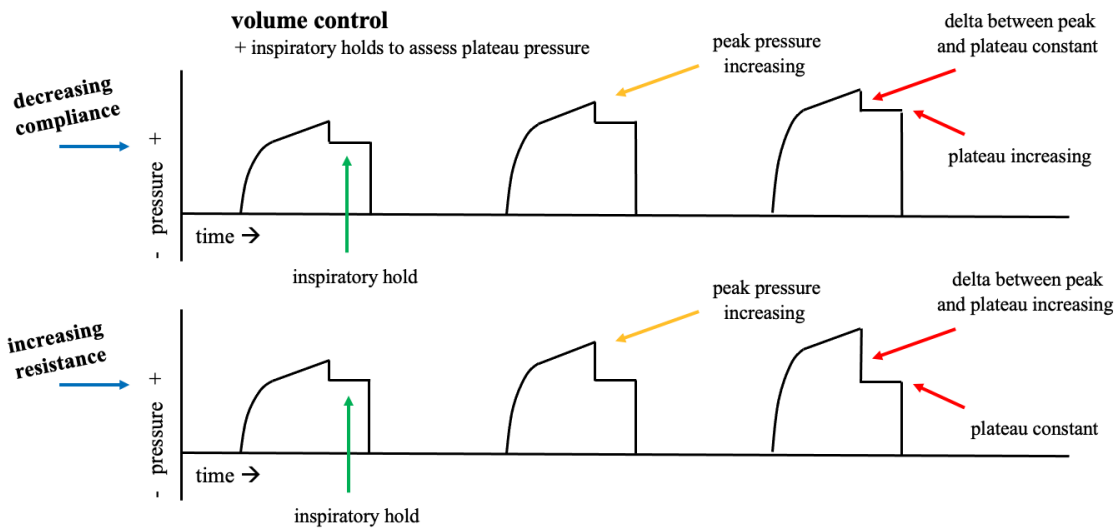
And then for pressure control:



An increase in **Resistance** is a similar example. The pressure waveform in volume control would be more or less the same; the flow waveform in pressure control would have a slightly different contour:[404]



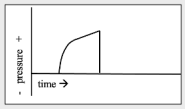
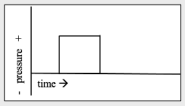
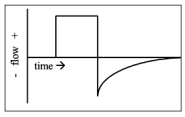
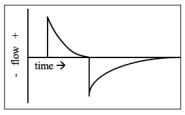
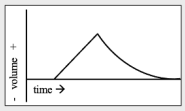
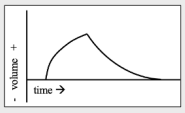
The recommendation here would be to utilize waveforms along with other parameters the vent measures and calculates. For volume control we would have:[405]



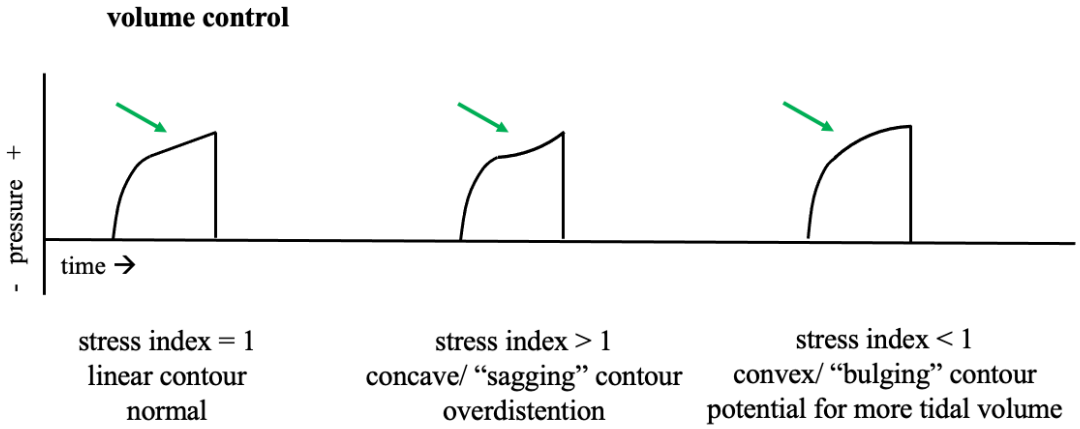
And in pressure control we would need to look at quantified or measured value for compliance and resistance.

[404] Hess, 2014 - The difference between these two flow waveforms in pressure control is spelled out in this paper
[405] This was discussed quantitatively/ without **Waveforms** in **Watching Pressures**

Moving on and summarizing: we can then sort these things and the simple observations we discussed above into a chart that outlines where we might look for evidence of certain things, depending on how we are ventilating the patient:

regardless of control	volume control/ fixed flow	pressure control/ decelerating flow
pressure waveform - (potential space for ventilation)	 dependent, look here! - pt effort - flow starvation - compliance change - resistance change - stress index/ contour	 controlled - (leak)
flow waveform - potential space for ventilation - AutoPEEP	 controlled	 dependent, look here! - pt effort - flow starvation - compliance change - resistance change
volume waveform - leak - (AutoPEEP) - (potential space for ventilation)	 controlled	 dependent, look here! - pt effort - flow starvation - compliance change - resistance change

A few notes on the interpretation of this chart. First is that there is one concept we have not yet discussed. If we have a patient in volume control (i.e., when flow is fixed during inspiration), we can look at the pressure waveform and evaluate if it is linear or if it curves in either direction. We could theoretically quantify this change in pressure as it relates to compliance (and call it "stress index"), then use that number to decide if we are optimally recruited, overdistended, or under-recruited (i.e., we can safely increase tidal volume).[406] It may make more sense when sketched out:



Another thing to note in the chart is that there are some things that can be seen or appreciated in multiple places. The primary places to look are noted in plain text, alternate locations are noted with parentheses.

One other thing to mention is that this discussion and the chart presented should not lead us to think that just because we are in a certain control of ventilation we will never see changes to the waveforms of controlled parameters. As we'll see in the very next section, **Asynchrony & Dyssynchrony** can manifest across all waveforms, regardless of control. That said, it helps to focus on the dependent variables/ waveforms, as that is where most information can be gleaned.

[406] Hess, 2014 - Refer here for a more detailed explanation of this concept of stress index (was also noted above, in reference to how resistance shows on waveforms depending on **Control of Ventilation**)

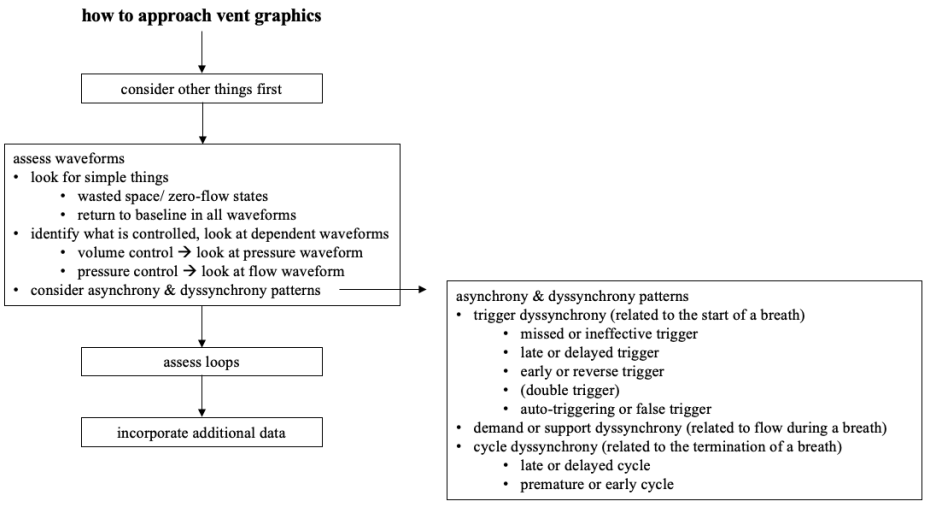
And then recognize that as we introduce loops there will be even more ways to identify some of these issues. Our recommendation (as mentioned in the introduction to this chapter) is to start with things the vent tells us right away (i.e., peak inspiratory pressure is almost always present on the vent home screen unless we intentionally take it away), then look at waveforms even if it's something we could find in a back menu or by performing a maneuver (i.e., measured positive end-expiratory pressure and AutoPEEP), after that consider moving on to loops if they are available. We'll summarize the workflow later, but it may help to keep this in mind, as many pieces of information can be gleaned from multiple sources.

Asynchrony & Dyssynchrony

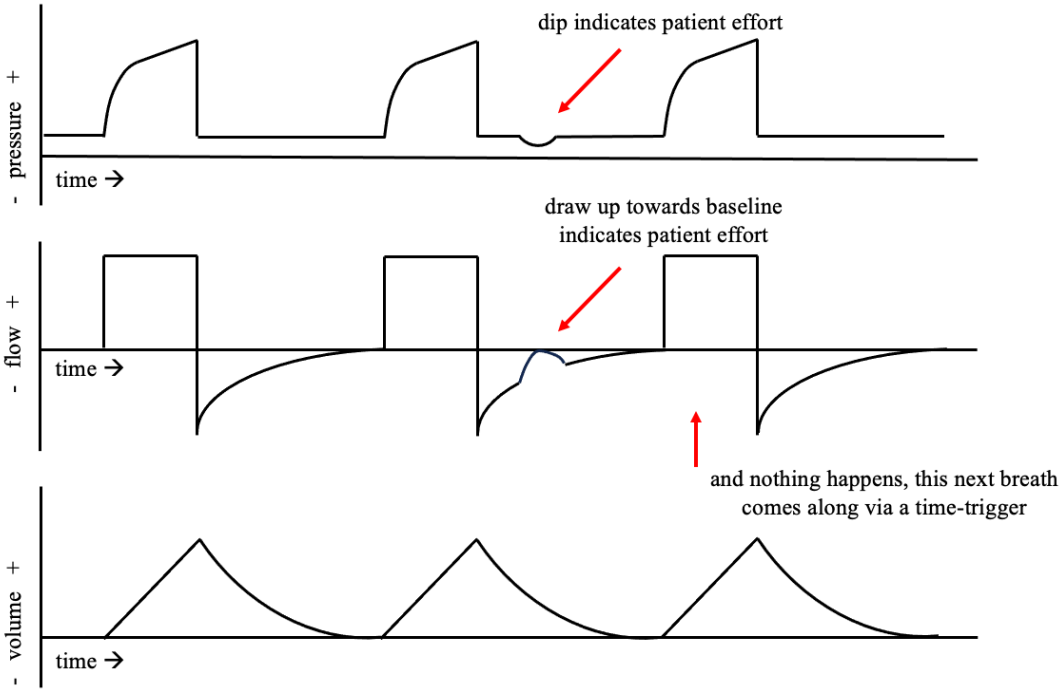
Asynchrony and dyssynchrony are terms in mechanical ventilation to describe situations in which patient effort and ventilator effort are misaligned or mismatched in a way that makes the therapy less useful or even harmful. The two terms represent a spectrum: a little out of synch would fall under dyssynchrony, completely out of synch would be asynchrony. Moving forward we'll stick with dyssynchrony as a general term, but just know that if things get really dyssynchronous or if dyssynchrony is really bad, we're more likely in asynchrony territory.

All of the **Three Big Things** are negatively affected by dyssynchrony, so it is helpful to be able to recognize them. More specifically, **Comfort** or discomfort/ lack of comfort is often related to dyssynchrony. When we first introduced the idea we focused mostly on medications (both analgesics and sedatives), but mentioned that sometimes we can make subtle changes in our vent settings to optimize comfort. **Waveforms** allow us to zoom in on this idea and identify specific patterns. Which then allows us to make precise changes to fix whatever problem we have, versus simply trying things and waiting to see what happens.

Before diving right into more waveforms and identifying all the patterns of different dyssynchronies, let's start with a general framework. We can have dyssynchrony in three different general areas: at the start of the breath (i.e., issues with **Triggers**), during the breath (i.e., issues with **Flow**) and at the end of the breath (i.e., issues with cycle or termination). And then within some of these general categories, we have different things that can go wrong. Let's just start with an overall list, and then we'll work through them.[407] And note that some of these are things we've touched on already.



Starting at the top, we have a missed or ineffective or failed trigger. In this type of dyssynchrony, we see evidence of patient effort in the pressure and flow waveforms, but the machine does not deliver a breath:[408]



Most common causes would be a **Trigger** set too high or the presence of **AutoPEEP**. Fixes, therefore, would be to check for or rule out AutoPEEP (and fix if necessary), then decrease the trigger sensitivity.

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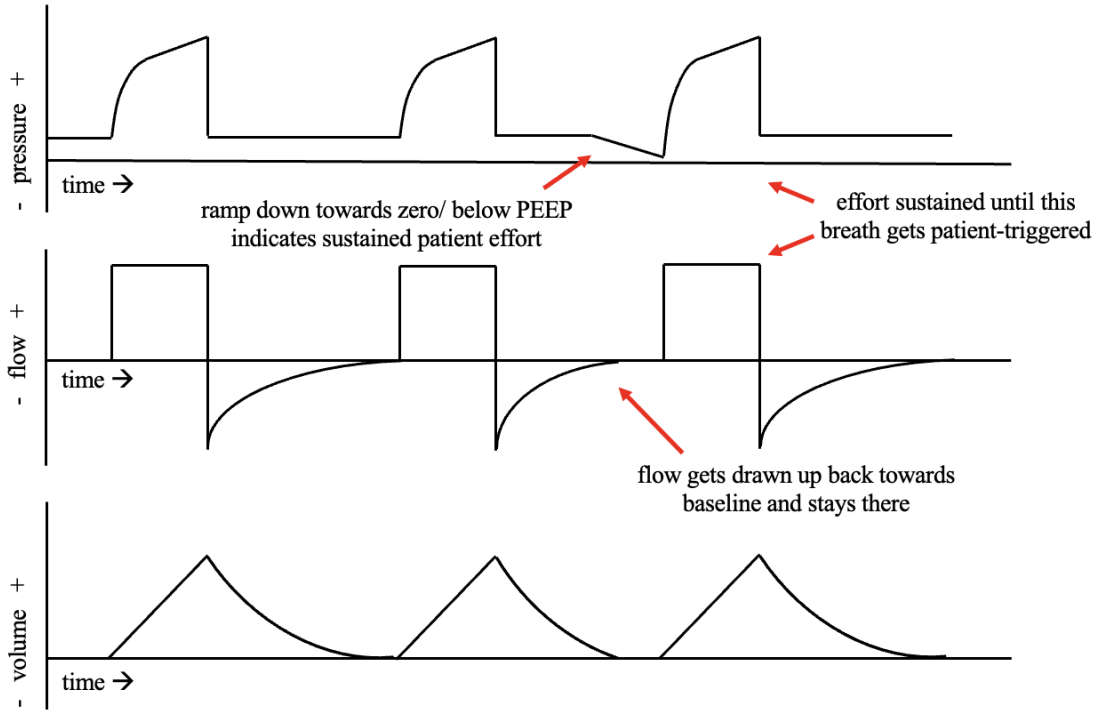
[407] Bishop, 2025; Ramirez & Arrelano, 2017 - This list we have put together is similar to those presented by both Melody Bishop in her video series on Instagram and this paper from Ramirez & Arrelano; there is a bit of a difference, however, and we'll discuss those along the way

[408] We'll draw these out in volume control just for consistency; unless otherwise mentioned or demonstrated, the dyssynchronies will look similar in pressure control

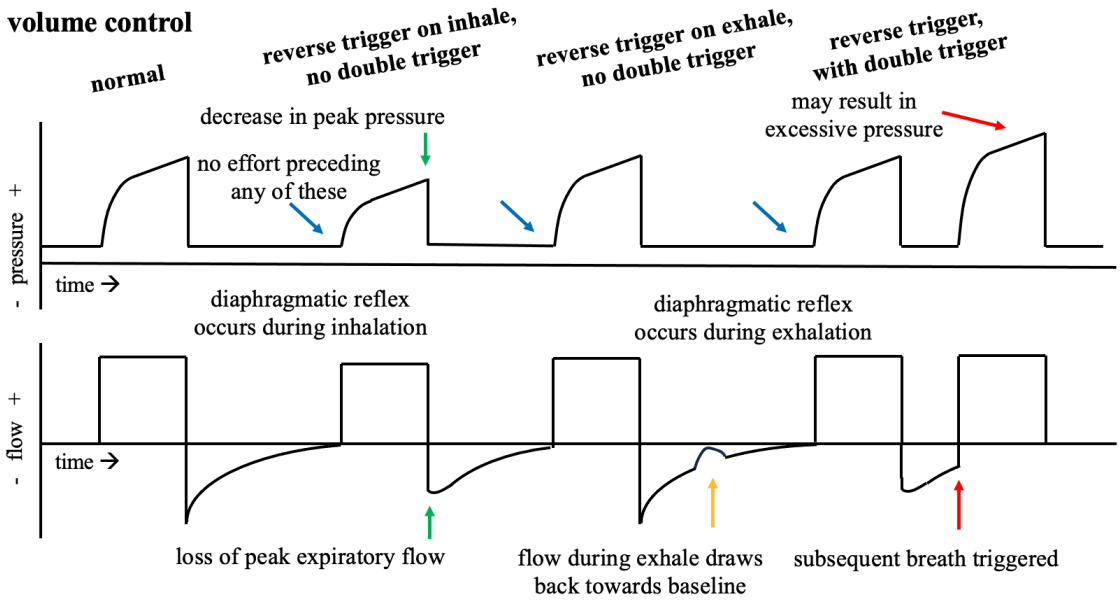
Next is a late or delayed trigger.[409] This one occurs when the patient is trying to take a breath, that effort goes unreciprocated by the vent (i.e., no breath delivered), but then the patient continues to pull harder until he or she does, in fact, meet the trigger threshold. It looks like this:

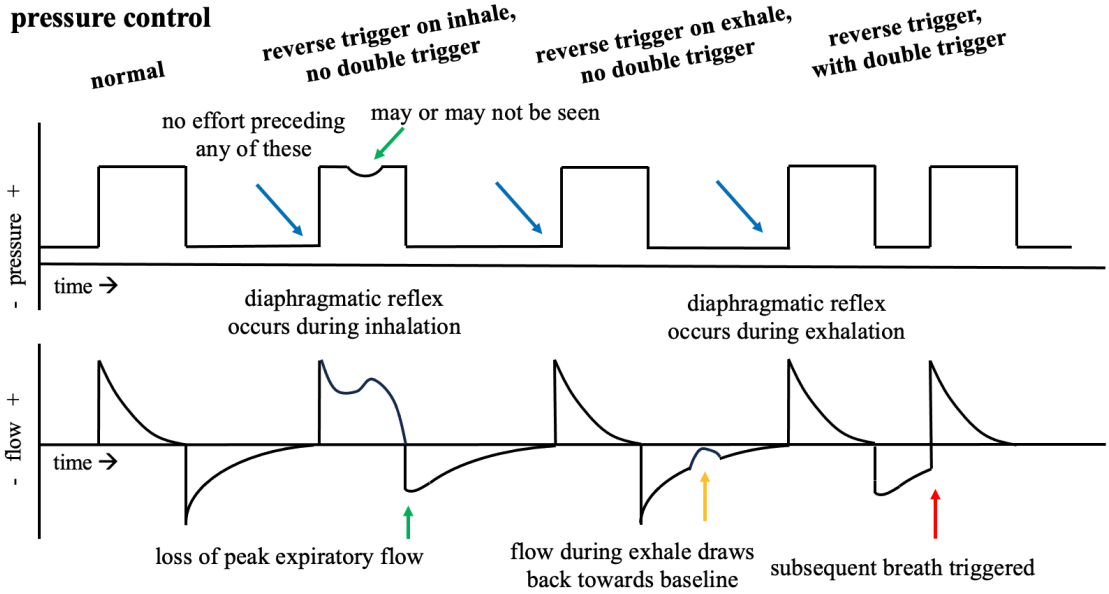


Fixes, just as above with a missed trigger, would be to eliminate AutoPEEP and then consider decreasing the trigger sensitivity.

[409] Mireles-Cabodevila & friends, 2021 - The article we just mentioned in the last footnote doesn't actually cover this one, but it is discussed here in a much more detailed and comprehensive overview of all things dyssynchrony

After that we have the early or reverse trigger. In this situation, a machine-delivered breath is followed immediately by patient effort. Basically, the machine-delivered breath causes the patient to make this effort (versus the normal in which the patient triggers the machine, hence the name "reverse"). To say this another way, passive inflation from a time-triggered and machine-delivered breath causes contraction of the diaphragm.[410] Now this diaphragmatic contraction can happen at different points along the breath and it may or may not trigger a breath, but whichever way it plays out it can be seen on the waveforms.[411]





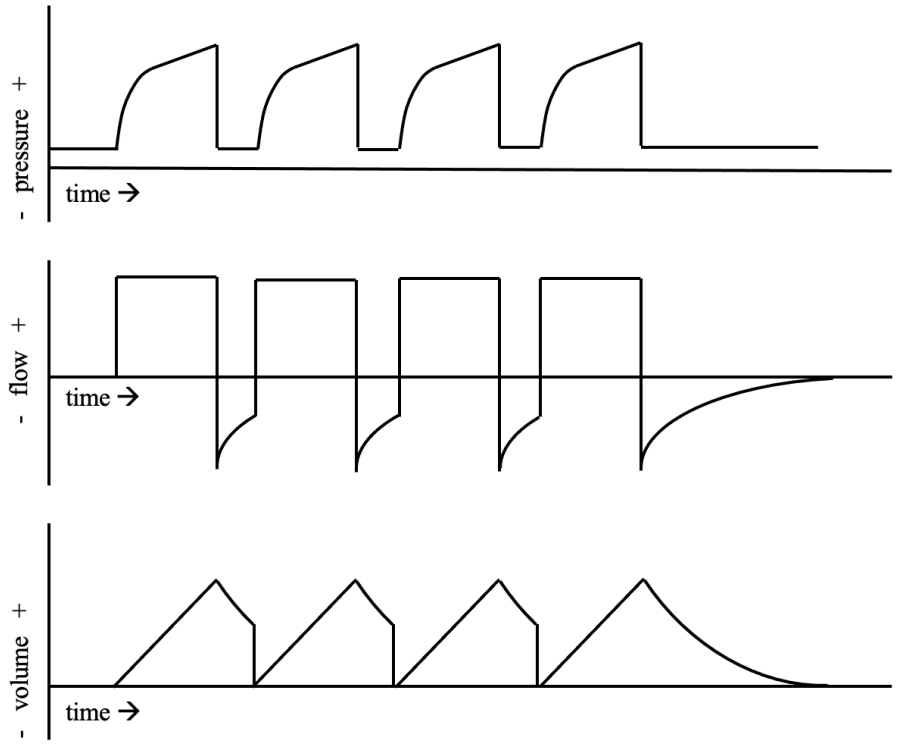
The most likely cause for this one is oversedation, therefore the fix would be to allow the patient to wake up just a bit. It could also be the case that decreasing tidal volume or rate to reduce stimuli to the

[410] Núñez Silveira & friends, 2023 - Learn all about the reverse trigger in this review paper
[411] And if a breath is, in fact, triggered - this is that "medium-risk" situation we notated in yellow way back in the section on **Control of Ventilation** when we first introduced the idea of **Assist Control**

diaphragm could relieve this.[412] Then there is the possibility that this sort of thing can be caused by a severe brain injury and in that case it may be necessary to paralyze the patient in order to ventilate effectively.

The next issue to discuss is more of a semantic one, but it warrants discussion because it gets used quite often. A double trigger refers to the idea of two full breaths being given back to back. Looking back at the reverse trigger situation we just discussed, if a breath does get triggered due to the reverse trigger dyssynchrony, that might get labeled as a double trigger. The issue with that, however, is that there are other things that can cause two full breaths to occur one immediately after the other: reverse trigger, early cycle and auto-triggering. We'll discuss the latter two concepts in turn, but the takeaway here is that if we see double triggering, we need to investigate further.

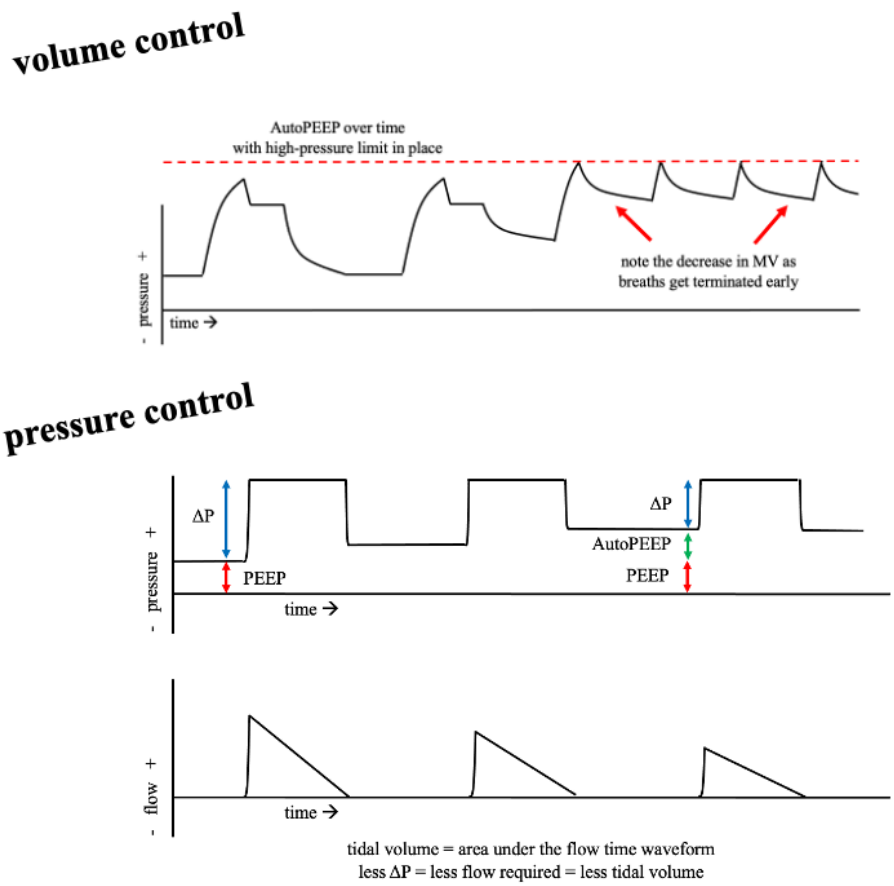
The last trigger dyssynchrony is auto-triggering or the false trigger. We discussed this idea already when we discussed **Triggers** initially, so we'll skip the explanation and simply show how it might look on the vent screen. Also note that what we see varies a bit depending on the cause of the auto-trigger or false trigger. First is how it looks if we auto-trigger a handful of breaths in succession:[413]



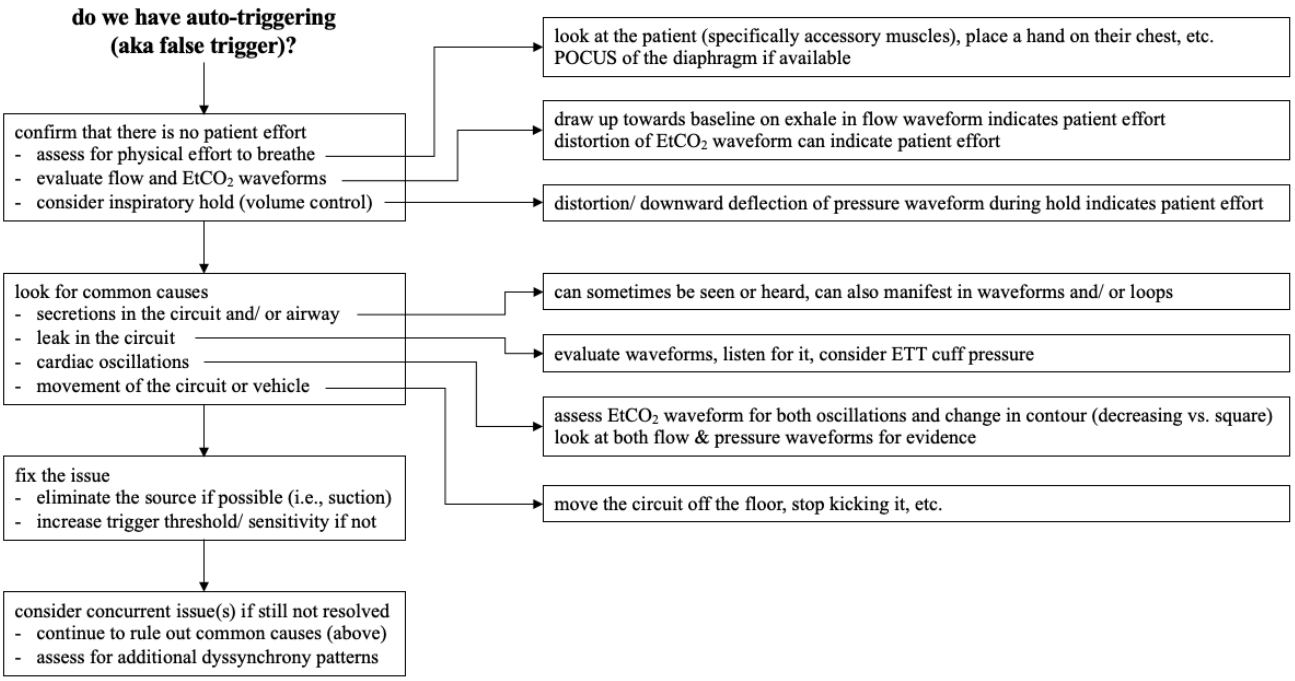
[412] WhiteboardMedicine, 2025 - We'll cite this video again later, but it does a good job of explaining reverse trigger and early cycle (although it refers to early cycle as double triggering, which is different than how we present it here)

[413] Note that this is different than **Overbreathing** in which there is a physiology reason for an increase in patient's physiologic rate of breathing

Recall that the issue here is that we can lose minute volume precipitously if this is sustained (due to breath stacking and cumulative AutoPEEP):

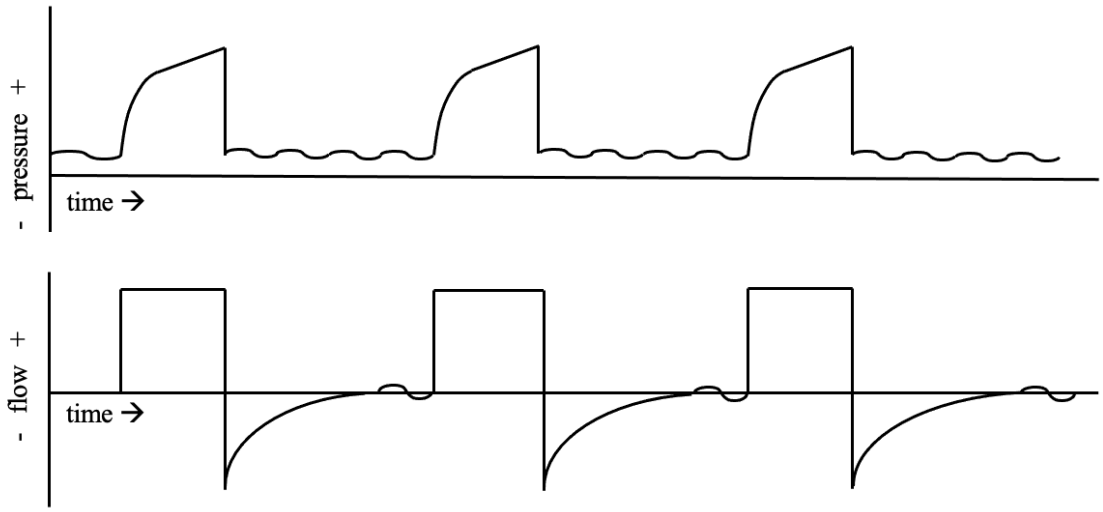


With suspicion of auto-triggering, consider an algorithmic approach. Confirm that there is no patient effort (i.e., that we do, in fact, have a false trigger), then eliminate the potential cause:



One thing mentioned above that isn't demonstrated elsewhere is the idea of cardiac oscillations.[414] These could be due to the patient's heartbeat (hence the name), but also could be caused by pulsatile flow in any mechanical device (like a hemodialysis machine or a cardiac device such as an LVAD or IABP). Basically the oscillations or movement of whatever is the culprit (the patient's heart or an external device) create enough of a change in flow (or pressure, depending on how the trigger is set) that breaths are falsely triggered. This will likely present as shown above with multiple breaths in sequence.

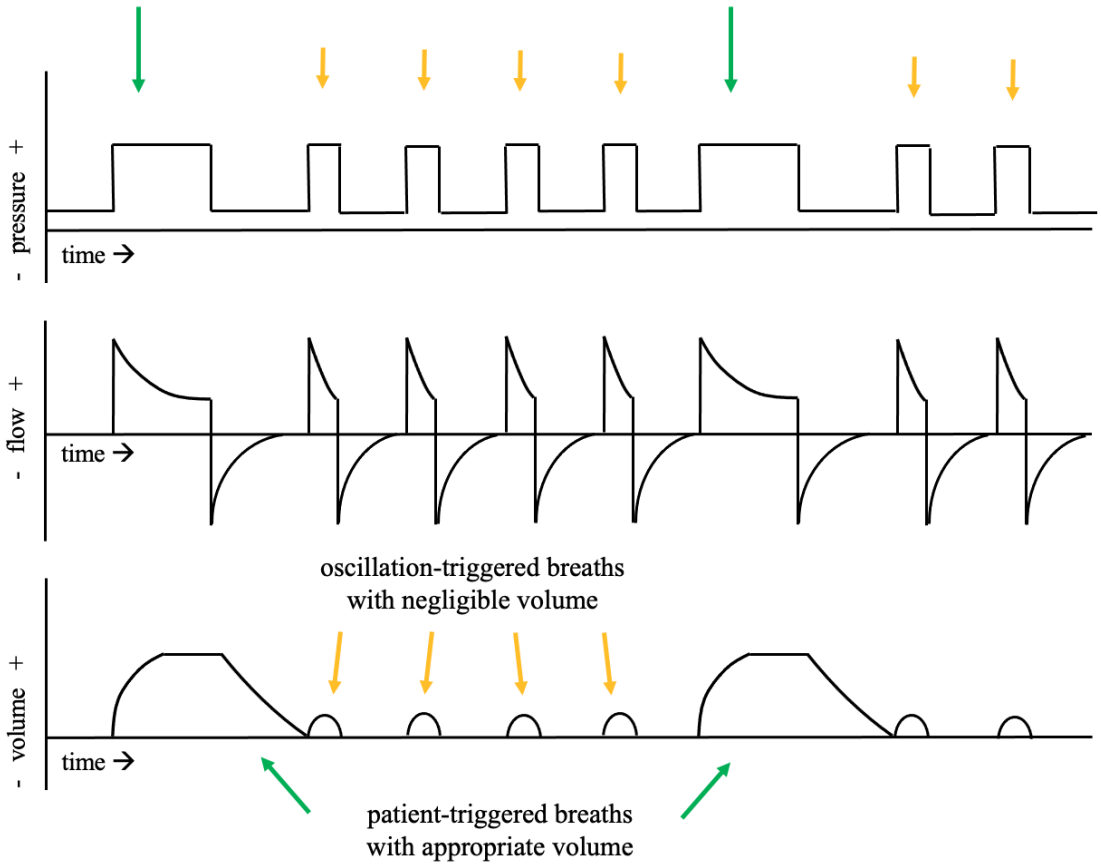
Now if oscillations are present and below the trigger threshold, they can sometimes be seen in the pressure and flow waveforms:[415]



[414] Bishop, 2025 - Everything we mention on this specific subject was taught to us by Melody Bishop, RT
[415] Mireles-Cabodevila & friends, 2021 - Or even in the end-tidal waveform, as mentioned and shown here

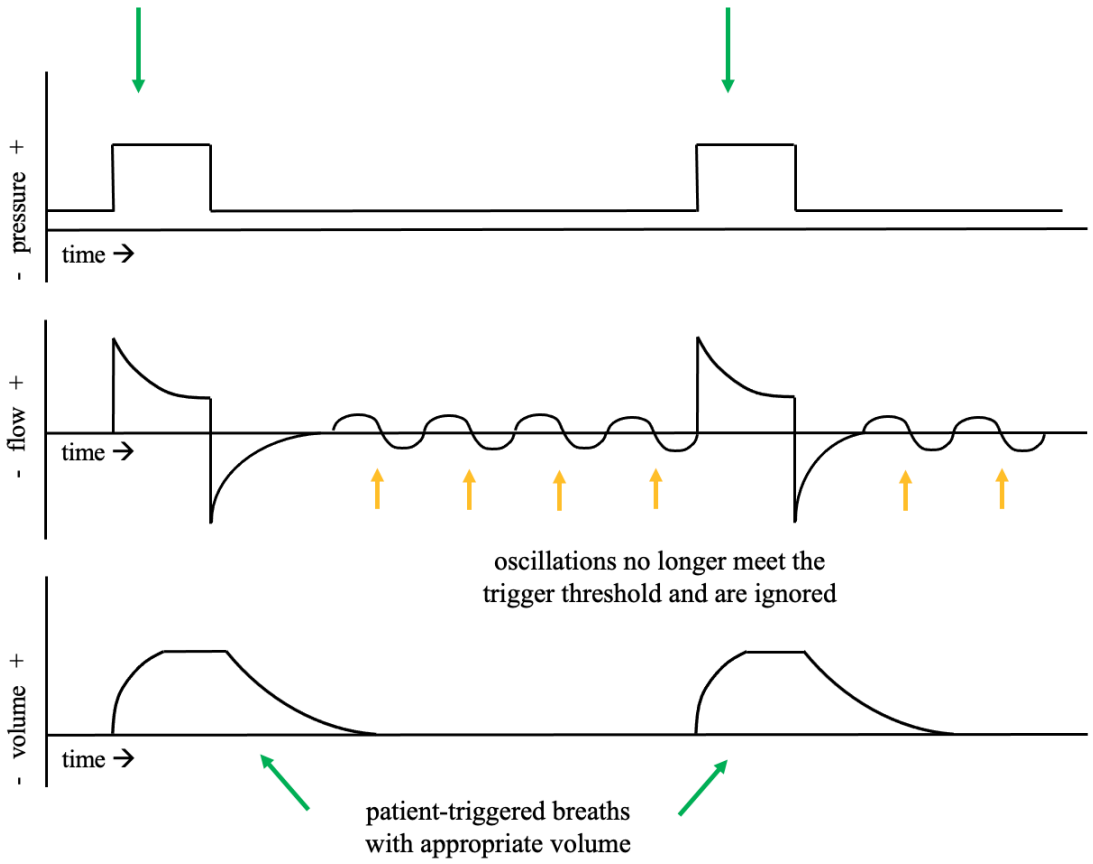
That said, this will be very tough to see on the pressure waveform and there may not be any zero-flow time in the flow waveform for this to happen.

And then if the oscillations are at or above the trigger threshold, we will simply see a series of stacked or auto-triggered breaths, one right after the next. If we see this and want to know if it is, in fact, due to cardiac oscillations (versus some other cause), consider swapping to a mode of ventilation that includes pressure support breaths.[416] We will then be able to see the difference between breaths that coincide with patient effort and ones that are falsely triggered by oscillations:

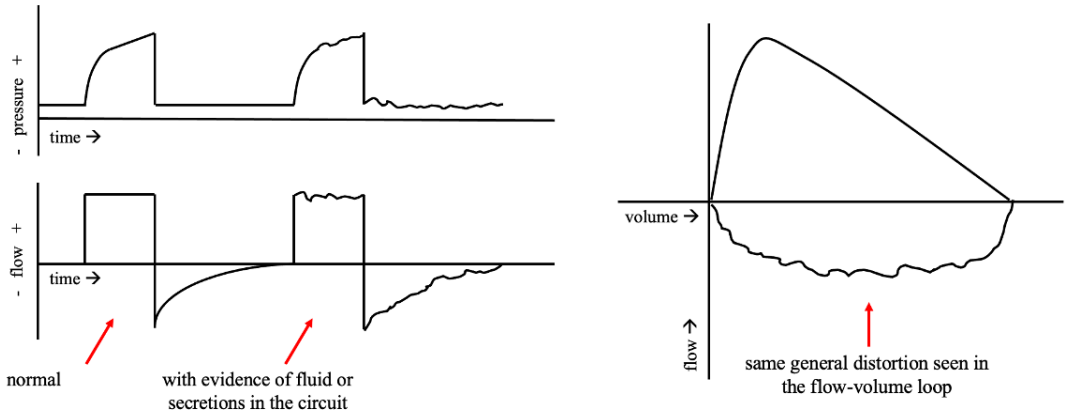


[416] The reason for this (swapping to pressure support or even **Synchronized Intermittent Mandatory Ventilation**) is that if we have auto-triggering of breaths that are assisted (versus supported) we will either end up with very high pressures or drop **Minute Volume** as these breaths get terminated early

And then the fix is to increase our trigger sensitivity so that the oscillations no longer trigger breaths and we just have true patient-triggered breaths left:



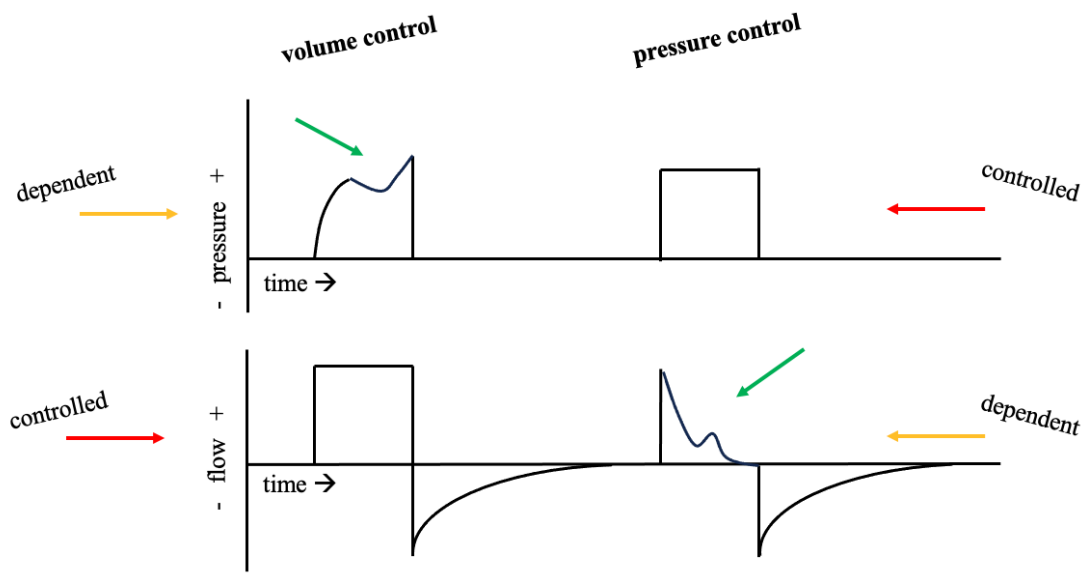
Another thing we haven't yet demonstrated is evidence of secretions or water in the circuit. It's looks like small-wave distortion and can be seen in various places (including **Loops**, which we'll get to soon):



And then just keep in mind that we sometimes see multiple dyssynchronies happening simultaneously. So we could, in theory, have a patient that is auto-triggering in addition to another issue. Which is problematic because we generally want to rule out patient effort to know auto-triggering is happening, but it could be the

case that the cause of the auto-trigger simply is stronger than the patient's own effort to breath. The solution here would be to assume the auto-triggering or false trigger, fix what we can to alleviate that as a possibility, then continue to assess the waveforms for further dyssynchrony. We just need to ensure that we avoid that low minute volume state that can occur with repeated auto-triggering.

So that's all of the trigger dyssynchronies. Next is the one dyssynchrony we have that applies to the middle part of the breath. It goes by many names: flow hunger (as we noted in **Waveforms**), flow starvation (used more often at the extreme end of the spectrum), demand dyssynchrony, support dyssynchrony and then most recently (and in an effort to avoid the issues inherent in all these other terms) work shifting.[417] As a quick review, since we discussed this already, let's take a look at the graphics again:



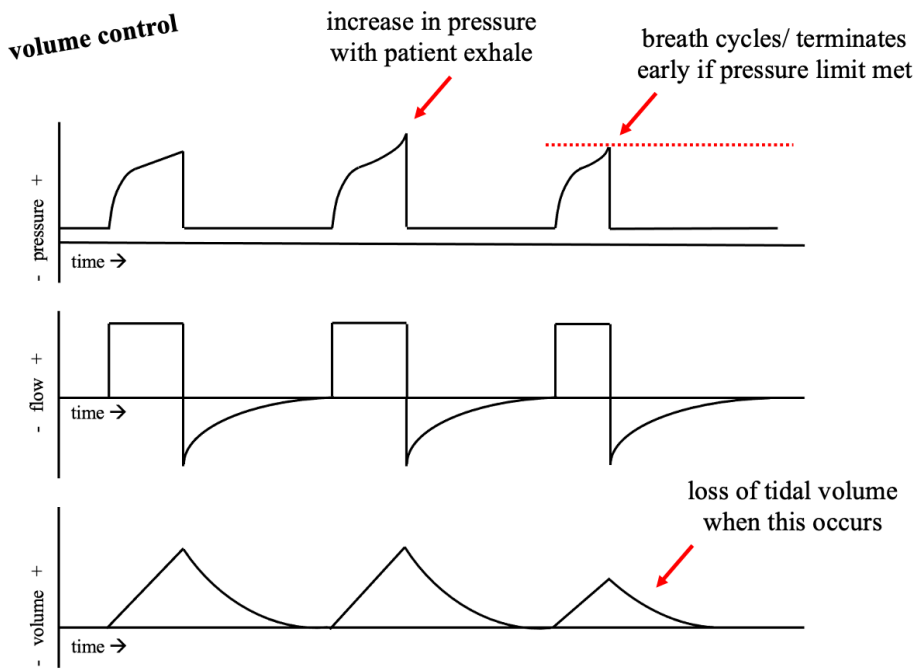
The fix in volume control would be to increase flow to meet patient demand. That said, we most often do that indirectly in transport by increasing tidal volume. In pressure control, we would increase peak pressure/ the pressure control parameter. And we do this incrementally until we see evidence of the demand go away.

As a sidenote on this topic of inadequate flow (which, again, goes by many names) - it is one of the more commonly observed ventilator dyssynchronies.[418] It is also relatively common to see this pattern concurrently with other dyssynchronies (i.e., flow hunger/ starvation along with an early cycle that results in double triggering).[419]

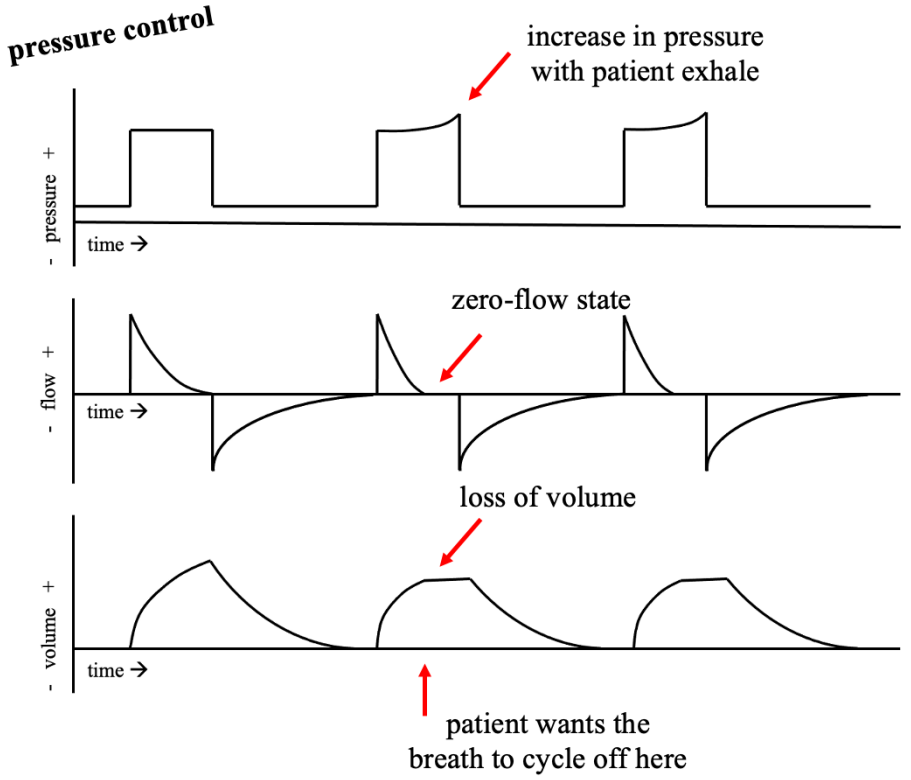
On next to our cycle dyssynchronies, beginning with late or delayed cycle.[420] This is when the patient starts to breathe out, but the machine continues to give the breath - the cycle from inhale to exhale is late or delayed. Think of it as the patient exhaling against the positive pressure of the breath being delivered. This will manifest in slightly different ways depending on control of ventilation.

[417] Mireles-Cabodevila & friends, 2021 - This idea of work shifting referring to the patient taking on work that the vent is failing to do; it is discussed at length here (along with all these other dyssynchronies as well)
[418] Scottile & friends, 2025 - This paper cites flow hunger/ starvation and reverse trigger as the most commonly observed dyssynchrony patterns (and then goes on to discuss implications and consequences)
[419] We'll come back to this shortly, just have to talk about cycle dyssynchronies first
[420] This term "cycle" has been mentioned many times along the way, but just for clarification: it refers to how the breath ends or terminates

In volume control we see an increase in pressure at the end of inspiration. And then we often will see that breath terminate early when our high-pressure limit it met:

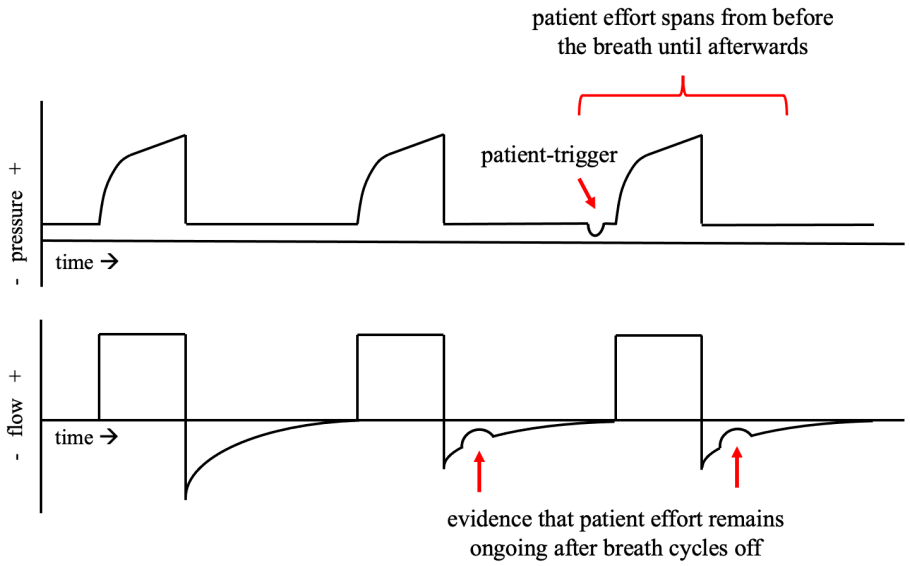


And then in pressure control we see a similar thing happen in the pressure waveform, but the rest is slightly different. First is that we have a drop in flow to a zero-flow state. We also see a flattening of the volume waveform with loss of volume. And we are unlikely to see the breath cycle off early due to a high-pressure limit being met:



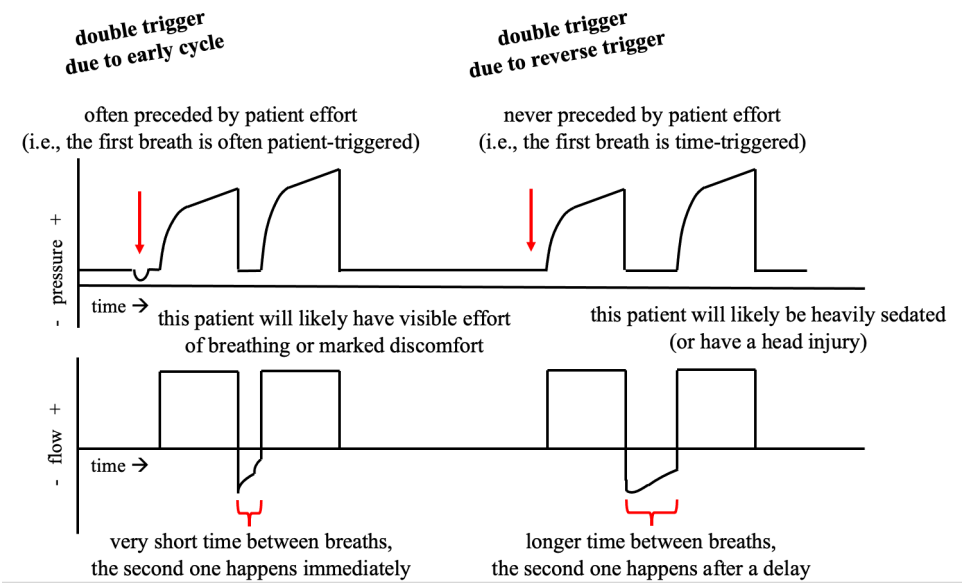
The solution here is to decrease inspiratory time or to adjust the termination trigger if it's a pressure support breath where this is noted.[421] Make small, incremental changes until the waveform starts to look normal again and be sure not to overshoot and end up with the next dyssynchrony on our list.

And then the next (and final) dyssynchrony is premature or early cycle. Which is when the machine swaps over from inhale to exhale before the patient is ready for it. We see this primarily as a distortion in the flow waveform - after the precipitous drop indicating that the machine has cycled the breath, there is a draw upwards (towards baseline) that indicates patient effort. This is often due to significant patient effort (potentially discomfort) and we may see that the breath is patient-triggered (which would indicate that patient effort begins before the breath and then extends until after the machine wants to stop it). Here's how it looks:



[421] Bishop, 2025 - And as a reminder, that would be in **Synchronized Intermittent Mandatory Ventilation** or in pressure support which we discuss in **Weaning & Pressure Support**

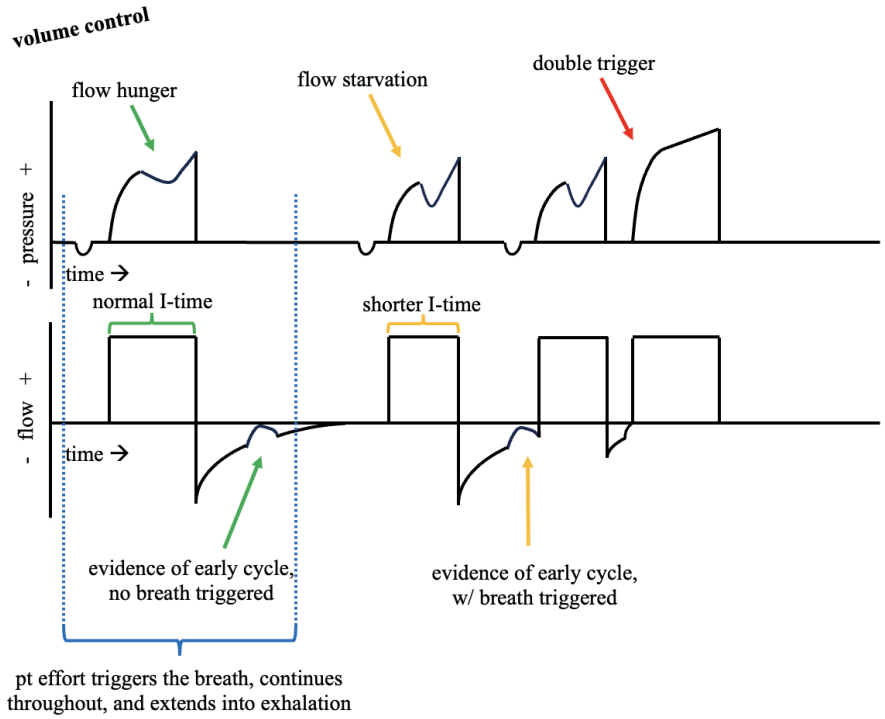
And this effort can, in fact, result in a subsequent breath being triggered. Which would be another case of a double trigger. To double-click on this idea: we mentioned before that a double trigger is not a precise term, as multiple things can cause two breaths to occur back to back. Here we differentiate a double trigger caused by early cycle (left) and one caused by a reverse trigger:[422]



If we have an early cycle dyssynchrony, regardless of whether or not it causes a subsequent breath to be triggered, the fix is to allow more time for inspiration or to increase inspiratory time. That or address **Comfort** with medications. And if we happen to see this in a pressure support breath, we adjust the termination trigger.[423]

[422] WhiteboardMedicine, 2025 - This video differentiates between early cycle and reverse trigger in detail (although it labels early cycle as double trigger)
[423] This idea is specific to pressure support breaths as discussed in **Synchronized Intermittent Mandatory Ventilation**

Very last thing to mention here is that we often see concurrent dyssynchrony patterns. One such example that we alluded to before is flow hunger/ starvation that gets worse and worse until it causes a double trigger due to early cycle:

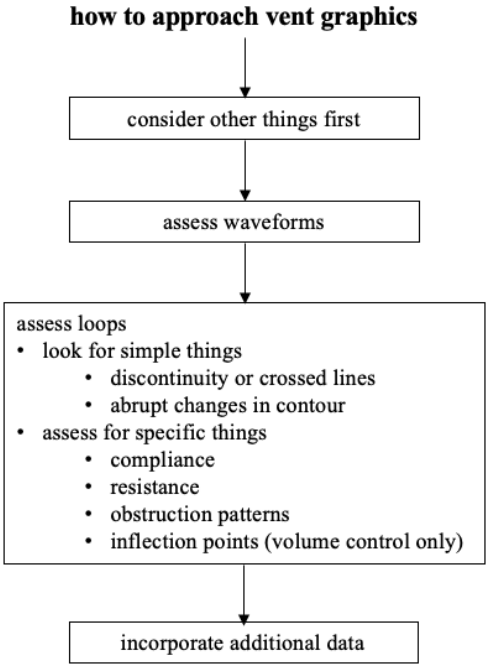


We'll review a step by step approach to working through all of this in **Utilizing Graphics in Vent Management** once we cover loops in this next section.

Loops[424]

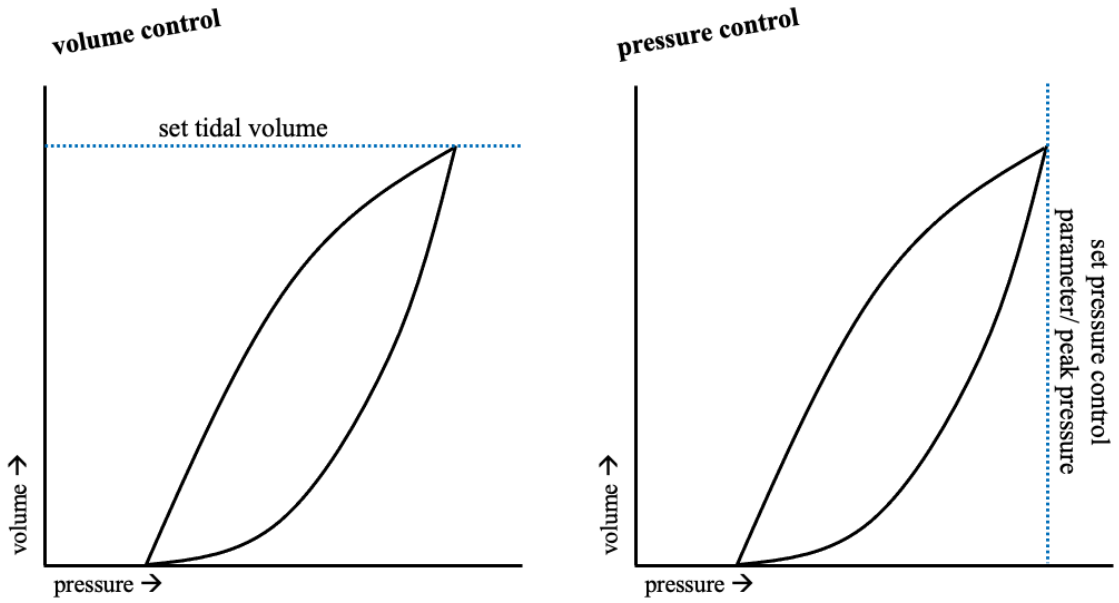
Next on our algorithm of understanding vent graphics are the loops. For the most part, most of the information we can gather from loops is also identifiable elsewhere, either quantified in measured/ calculated parameters or seen in the **Waveforms**. That said, some things are very easily recognized on loops and it is sometimes possible to adjust the vent screen to show waveforms and loops (in addition to the normal numbers) all at once. We can also trend changes and confirm clinical suspicion(s) by investigating loops and correlating to other findings.

As far as how we might approach loops, here's the overview:

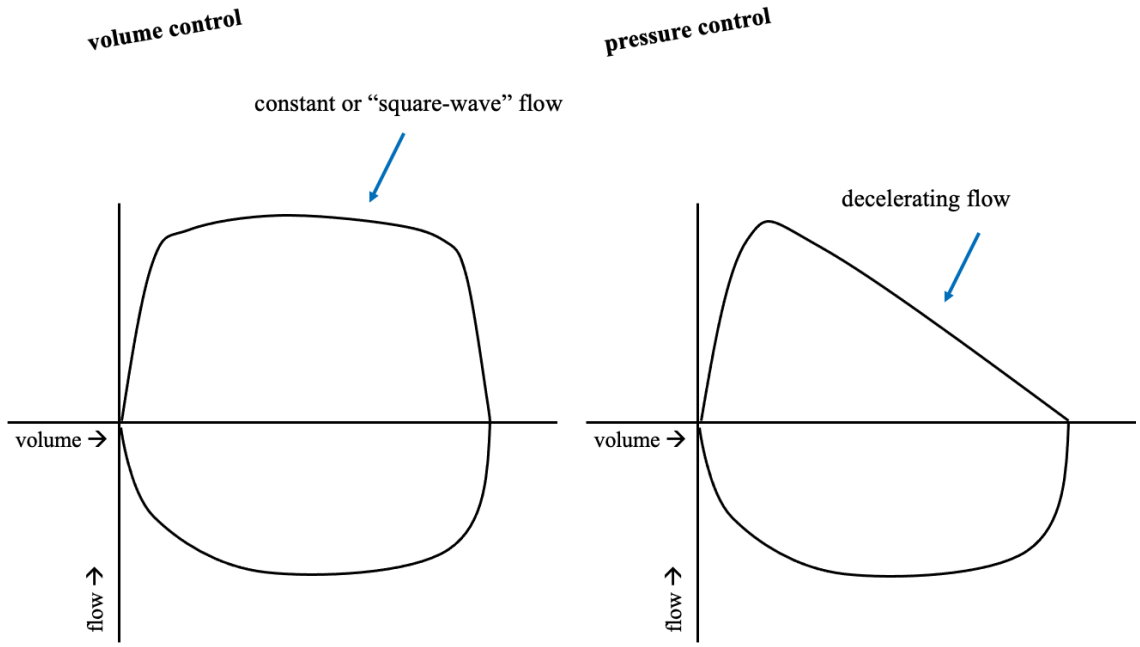


[424] Temlet, 2021; Hess, 2014 - Both provide a reasonable overview of loops and their utility in clinical practice

Let's just quickly review the normal loops. First is the pressure-volume loop. Note that depending on **Control of Ventilation**, the loop is limited on one end or one side by the independent or set variable:



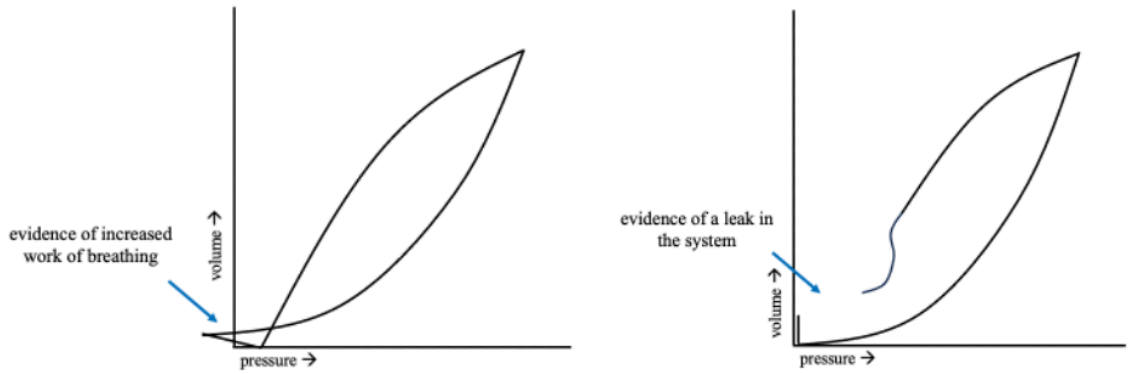
Next is the flow-volume loop that looks like this:



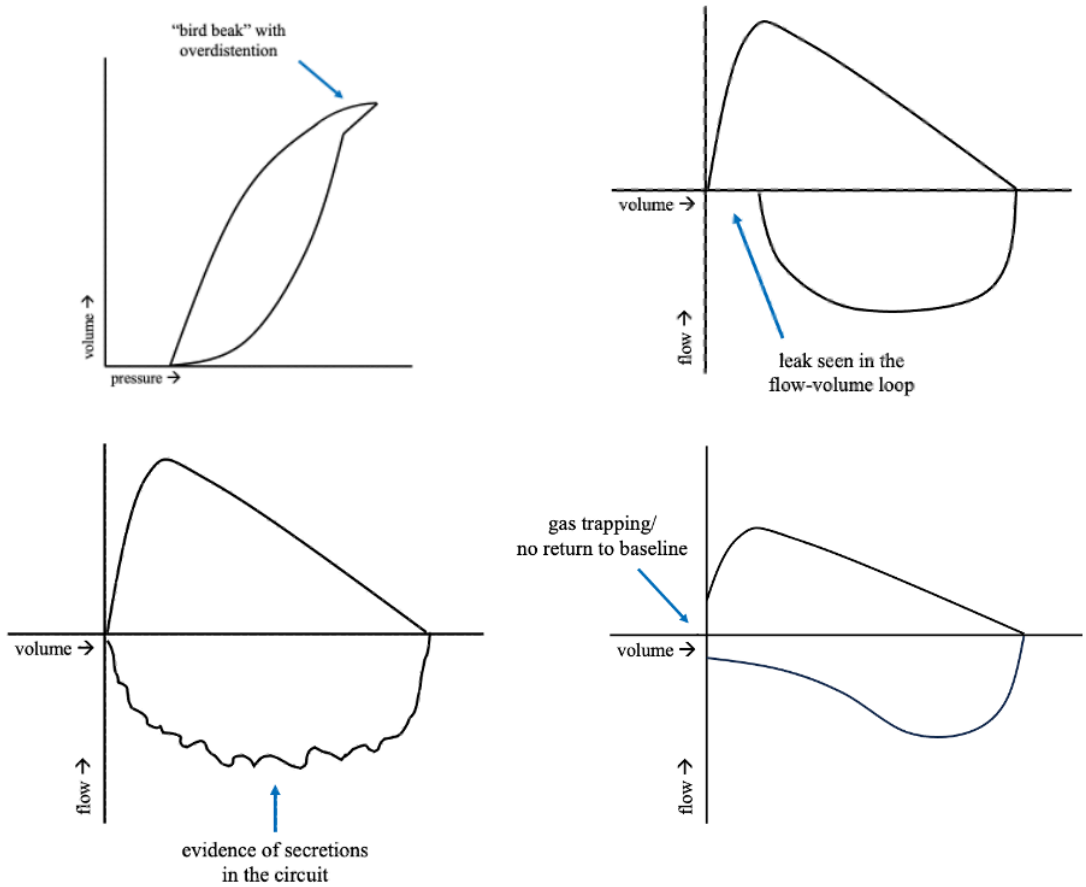
The pressure-flow or flow-pressure waveform is not typically used even though it is possible, but we'll skip over it in this discussion.[425]

[425] Devlieger & friends, 2002 - For those who are interested, however, this was the best overview we could find; it's specific to neonatal ventilation, but the general concepts apply to larger patients also

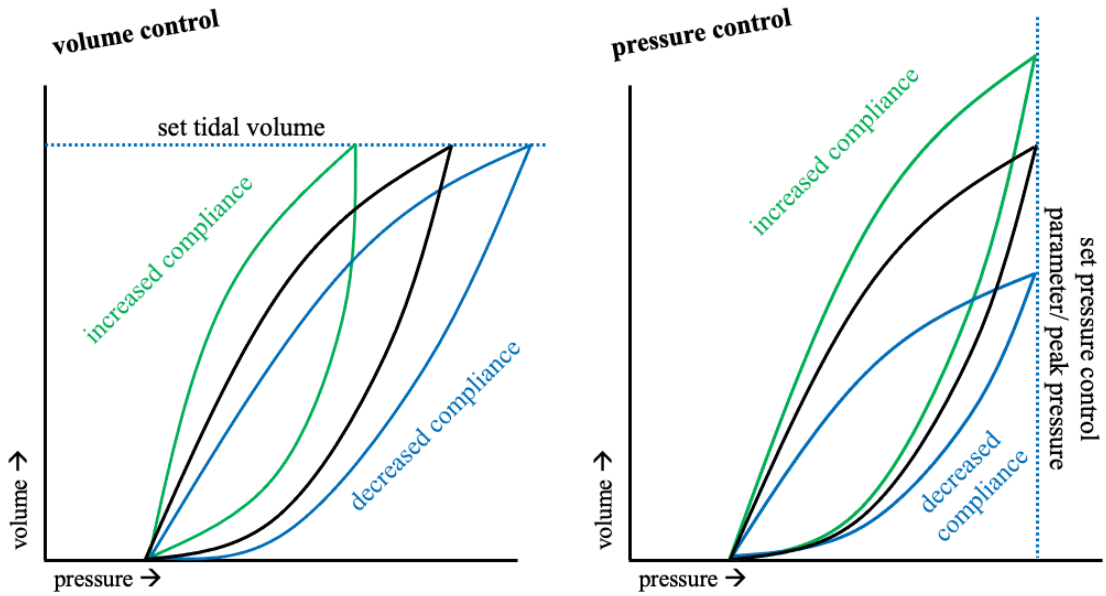
First thing to note is that all these loops have a sort of roundish shape. Things that break that shape can be readily identified and then fixed. Examples would be leaks or increased work of breathing by the patient, both seen in the pressure-volume loop:



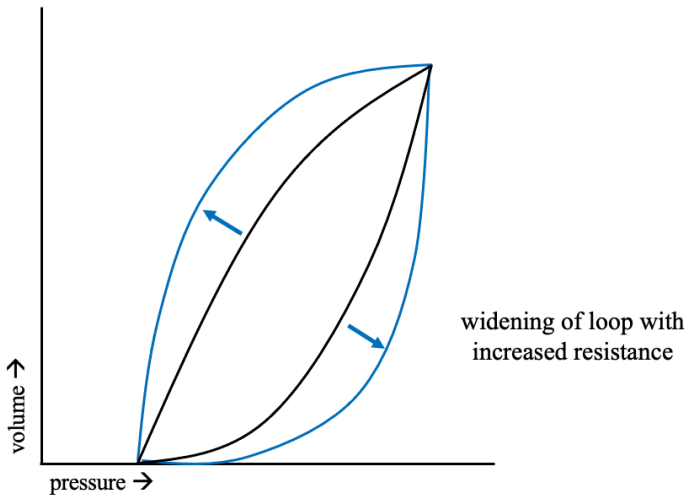
Another group of things that are easy to spot are abrupt or irregular changes in the contour of the loops. Things such as "bird beaking" with overdistention in the pressure-volume loop, a leak in the flow-volume loop, presence of significant secretions in the flow-volume loop, and/ or AutoPEEP in which flow in the flow-volume loop fails to return to baseline would fall into this category:



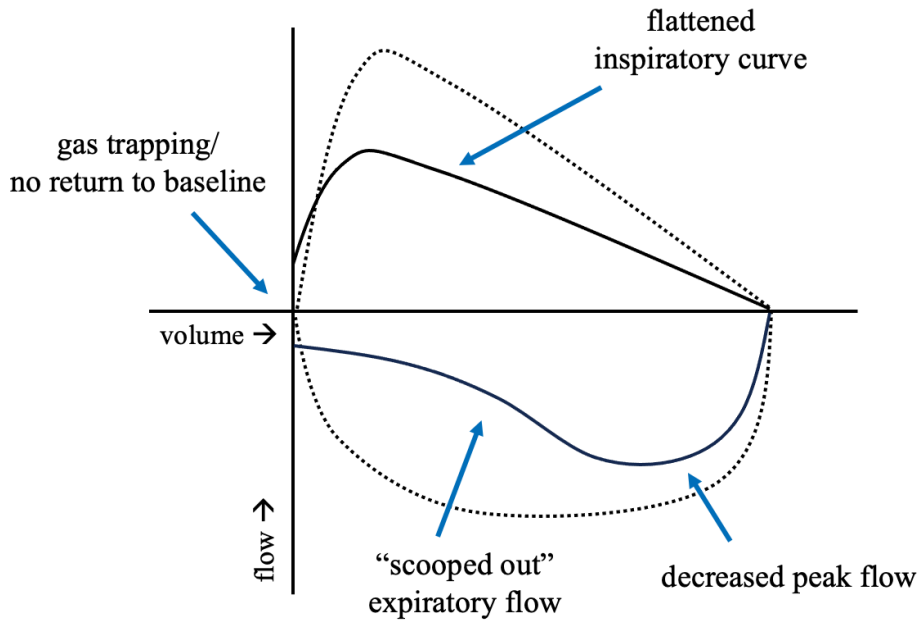
After checking for these sorts of things that we can see at a glance, we can then move on to assess a few specific things. First is to look at the overall tilt of the pressure-volume loop and its correlation to compliance:



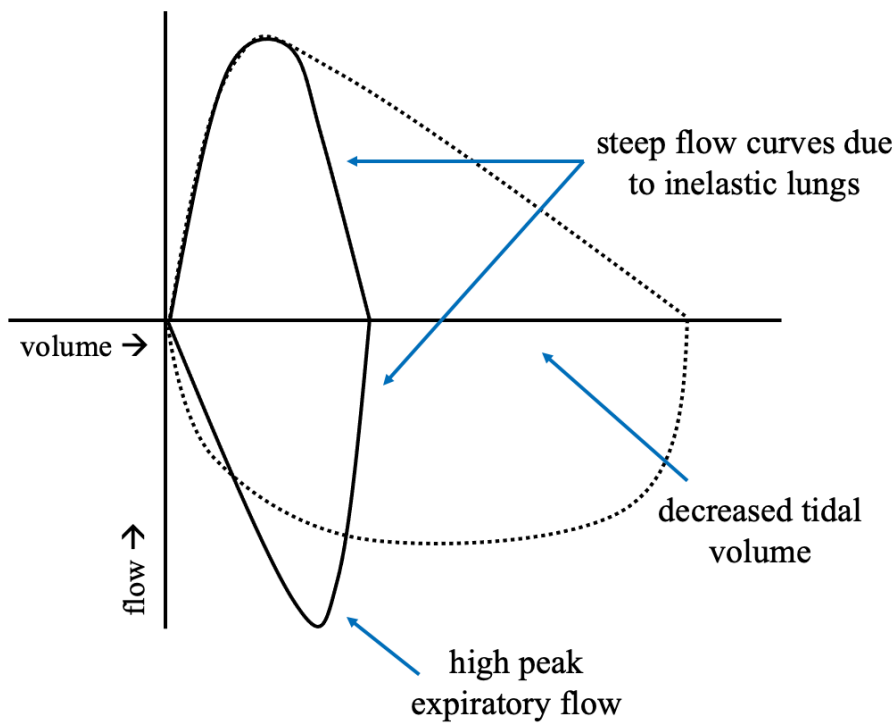
We also have the width of the pressure-volume waveform and its correlation to resistance:



Then if we look over at the flow-volume loop, we can evaluate for obstruction, bronchodilator response and/ or flow limitation on exhale:[426]

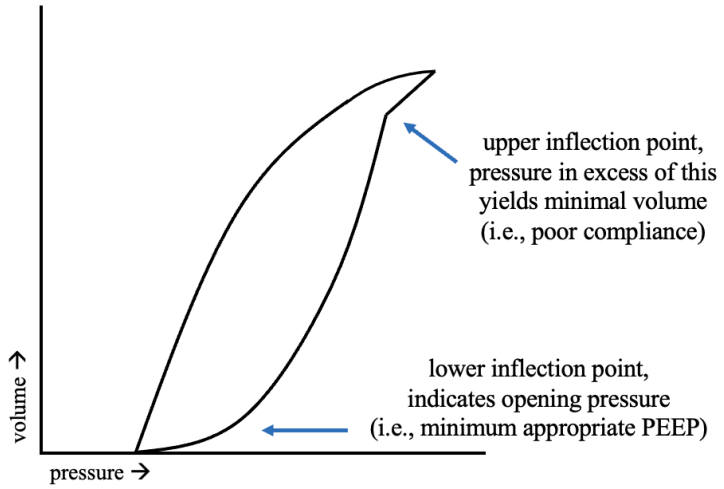


We may also be able to appreciate changes from restrictive airway disease (which also generally comes with poor lung compliance) in the flow-volume loop (as opposed to obstructive processes as shown above):



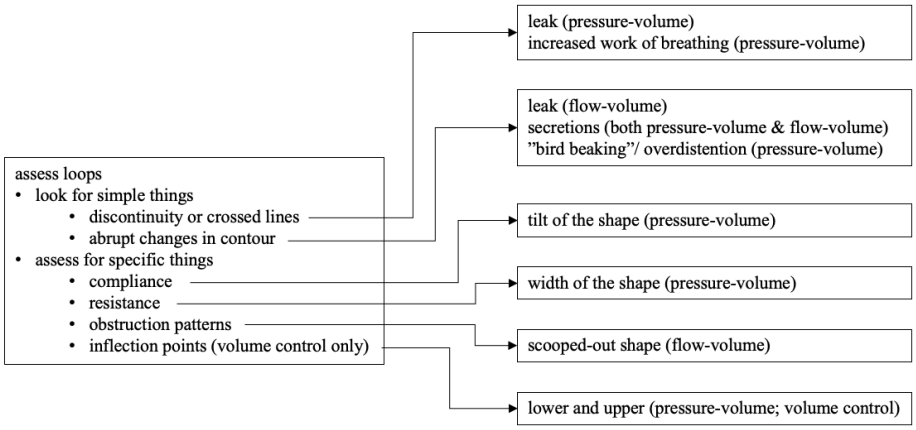
[426] Yartsev, 2025b - Both this and the next concept discussed here

And last thing to mention here is the idea of inflection points. This one is for the pressure-volume loop in volume control only. More specifically, when flow is held constant throughout a breath (i.e., a square-wave flow pattern), which is the case in volume control for most transport vents. Now the clinical utility of inflection points is a bit dubious, but we mention them here simply because they are often brought up in the discussion of vent graphics. We'll point them out in a graphic and then explain in detail:



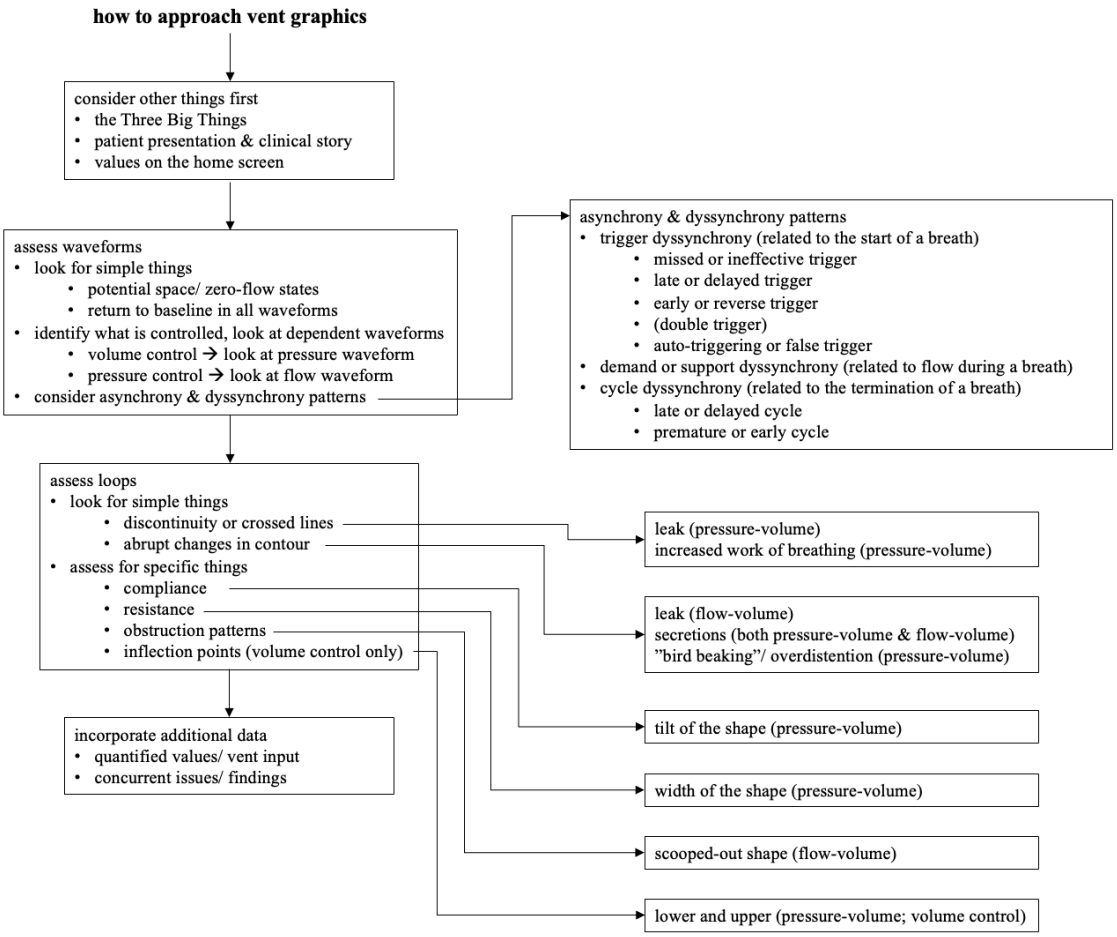
The idea is that the two inflection points provide guidance on appropriate pressures to target. The lower inflection point indicates a sort of opening pressure where compliance rapidly increases with alveolar recruitment and is often cited as the minimum appropriate value for positive end-expiratory pressure. The upper inflection point shows a decrease in compliance, beyond which additional increase in pressure provides minimal benefit.

And then we'll end with a quick summary on loops:



Utilizing Graphics in Vent Management

So here's what we have so far in terms of assessing vent graphics:



Recognizing that there is a ton of data in this flowchart and that many of the points require an understanding of certain concepts or the memorization of specific patterns, the next step is to discuss how we work through this algorithm and adjust interventions or vent settings based on what we see on the vent display. There are two general approaches to this sort of thing: a stepwise, algorithmic approach and pattern recognition.

In the stepwise approach, we simply start at the top of the flowchart (where it says "consider other things first") and work our way down. The advantage of this approach is that it is structured and easy to follow. The downsides are that it can take time and may not identify the most pertinent or likely threat first. An example of this would be a late or delayed cycle - if we approach the waveform interpretation in a stepwise fashion, we'd eventually figure out the issue after working through all of the other dyssynchrony patterns that are arbitrarily listed in front of this one. Which may be fine, but could be a disadvantage if time is of the essence.

Another strategy is pattern recognition. In this approach we simply get to know the various patterns and findings, then hope to spot them if they are present. Now this doesn't have to be a completely unstructured thing, rather we can utilize a strategy of looking at each graphic in turn - pressure waveform, flow waveform, volume waveform, pressure-volume loop, then flow-volume loop. This can help to streamline the process a bit, but avoids the top-down, systematic approach of looking for each thing one at a time.

A hybrid approach would be to work through the algorithm in broad strokes systematically (i.e., "consider other things first," then "assess waveforms," then "assess loops," etc.), but rely on pattern recognition

within each broad category. For example, maybe we try to commit all the **Asynchrony & Dyssynchrony** patterns to memory, but don't necessarily try to find each one in turn for every ventilated patient.

Regardless of how we choose to approach the thing, there are a few other things to mention about the algorithm. First is that we don't always need to go looking in the vent graphics for things to fix in the first place. Up at the top of the algorithm we have a reminder that some other things should take priority. If the **Three Big Things** are all sorted, there may not be a need to dig deeper. Likewise some clinical presentations will have expected abnormalities in the graphics that we don't necessarily need to go looking for - evidence of a leak with an uncuffed endotracheal tube in the pediatric patient or evidence of increased work of breathing in the DKA patient - these are things we should be aware of before looking at the graphics. Also some of the data that is commonly visible on the vent's home screen can assure us that all is well: peak pressure, minute volume, exhaled tidal volume and frequency. If these measured parameters match our expectations, then the need to go investigating further diminishes.

There are also some notes at the bottom of the algorithm under "incorporate additional data" that are worth reviewing. The "quantified values/ vent input" part refers to things that the vent can measure or calculate. Quantified values for **AutoPEEP**, **Compliance & Resistance**, etc., would fall into this category. Now we may choose to look at these things first, then refer to waveforms afterwards, and that's totally fine - it's more of a reminder that we can sometimes quantify the things we appreciate subjectively in the vent graphics.

And then the very last thing to mention is the "concurrent issues/ findings" part. It sometimes happens that we have multiple issues happening concurrently in the vent graphics (just as with our patients in general). This is particularly relevant when we are looking at **Waveforms** to evaluate for **Asynchrony & Dyssynchrony**. If we have evidence of multiple patterns or if patient effort makes it difficult to see certain aspects of a waveform, interpretation can be difficult. In these cases it may make sense to work through possible issues in a stepwise function and address each one as it is found. Also know that there is always the option to address **Comfort** pharmacologically[427] to ensure that **Oxygenation** and **Ventilation** are maintained - then as the patient begins to regain spontaneous effort or return to the prior state we may be better able to focus and/ or identify specific patterns and changes.

[427] See **Medications in Mechanical Ventilation**, specifically **Agents for Comfort and/ or Synchrony**

Additional Concepts Round Three

Expiratory Time Constant

Expiratory time constant (abbreviated RCexp) is a metric that describes how long it takes for exhalation to occur. It's a bit more complicated than just expiratory time though and to understand how it works requires a few steps. First thing is that we can conceptualize expiratory time constant as a descriptor of how the air pushed into the lungs on inhalation (by the ventilator) leaves the lungs on exhalation. Some lungs are very stiff or tight and, therefore, the lungs recoil quickly when inhalation cycle to exhalation; others are floppy or elastic and don't give much of a push on that body of air when it's time to breathe out. There's also the fact that some lungs communicate with the outside world via large openings (i.e., a patent 7.5 endotracheal tube) and others via smaller openings (narrower tubes, secretions, partial kink, extraneous devices, etc). This is all to say that while the expiratory time constant describes how much time is needed for the patient to breathe out, both **Compliance & Resistance** are involved.

Next thing with the expiratory time constant is that the value itself, notated in seconds, doesn't tell us how long it will take for exhale to occur, rather how long it will take for about 63% of exhalation to occur given whatever state of contributing factors as noted above. Which may seem a bit weird, but it's due to how the value is calculated via an exponential curve. That said, it is directionally true that a lower expiratory time constant means decreased compliance and/ or low resistance and that a higher expiratory time constant means increased compliance and/ or high resistance. But if we wanted to use that value to know how long expiratory time is or ought to be, there's a few more steps.

We now know that the expiratory time constant tells how long it will take for roughly 63% of exhale to occur in a given patient. Now if we go forward in time twice that, we're at 86%; thrice that 95%, etc. It looks like this:[428]

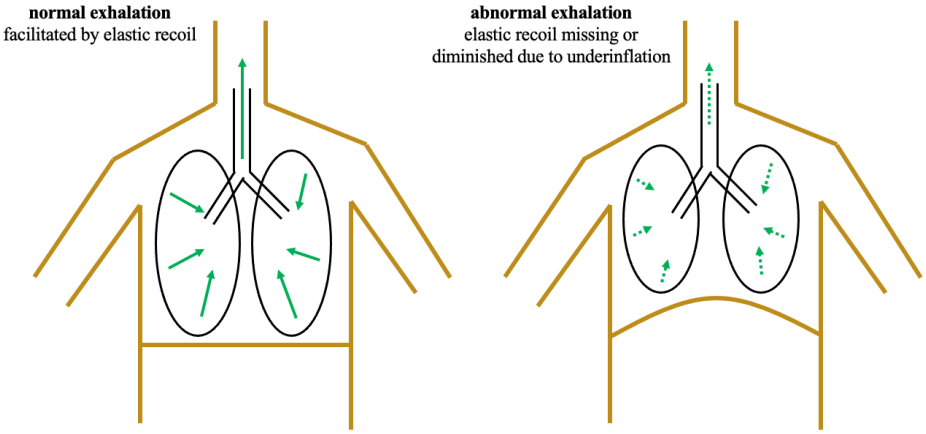
1 RCexp ≈ 63% exhaled
2 RCexp ≈ 86% exhaled
3 RCexp ≈ 95% exhaled
4 RCexp ≈ 98% exhaled
5 RCexp ≈ 99% exhaled

This means that we could hypothetically multiply the expiratory time constant by three or four or five and use that as exhalation time to ensure that exhale occurs to a certain degree. That said, this approach is less than ideal for a few reasons. First is that the goal of complete exhalation is related to avoiding **AutoPEEP** and there are ways we can evaluate for the presence of that directly, so no need to use a roundabout method.[429] Second is that the expiratory time constant value we get is variable and changes with patient effort, so while we may see a value of 0.5 and therefore assume we need a 1.5-2.0s expiratory time (for 95-98% complete exhalation), the patient can exhale forcefully to achieve full exhalation in less time that we might expect. And then lastly is that focusing too much on this one parameter can steer focus away from the **Three Big Things**, as we may end up losing **Minute Volume** at the expense of a "full exhale" that may or may not be clinically relevant.

One other big issue with using the expiratory time constant to determine appropriate expiratory time is that the value we see on the vent's screen is, in part, dependent on the settings we have dialed in on the

[428] Keller, 2017 - Quick and basic review of exponential functions and the idea of a time constant in general
[429] And this would be either an expiratory hold (as discussed previously in **AutoPEEP**) or simply looking at the flow waveform (as discussed just a moment ago in **Vent Graphics**)

machine. To make the point using extreme examples, let's say we have a patient ventilated with zero **Positive End-Expiratory Pressure** (or ZEEP)[430] or one who is being ventilated with low **Tidal Volume** in the absence of restrictive pathology.[431] In either of these cases, we may end up with an elevated RCexp because the lungs don't get filled as much as they normally would. The elastic recoil of the lungs that facilitates passive exhalation doesn't occur to the same degree if they don't fill up adequately:

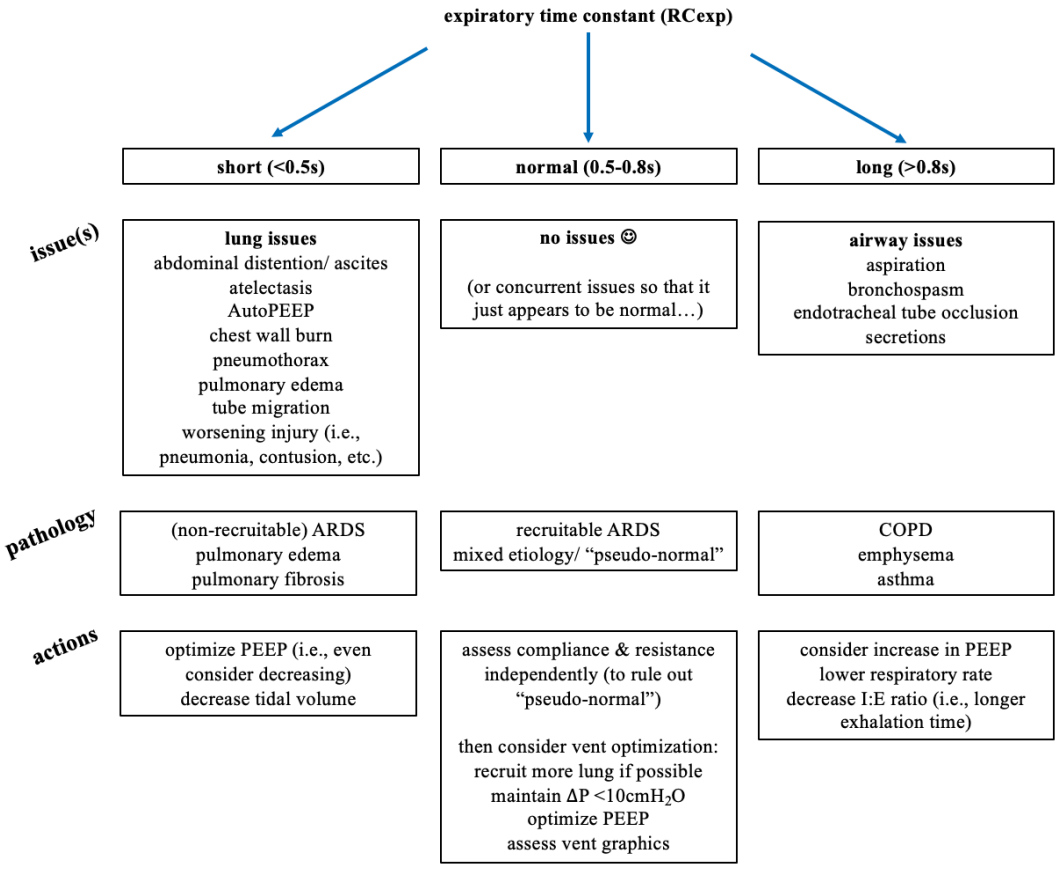


Now this isn't to say that the elevated or high value we see for expiratory time constant is wrong, per se, just that it is high or out of range due to things other than lung (patho)physiology. So while it is still true that it will take three times the expiratory time constant for 95% of the air to leave the lungs (assuming no patient effort), it's just that we could facilitate better exhalation by inflating the lungs more appropriately.

[430] This is pretty uncommon, but may be utilized in cases of **Hypotension** when there is marked concern for hemodynamic stability and limited access to other things

[431] This could be a patient who is markedly tachypneic and therefore **Tidal Volume** is decreased to maintain a normal **Minute Volume**

But let's say that we do have settings worked out appropriately and we'd like to see what value can be parsed from the expiratory time constant. Here's a quick reference chart and then we'll discuss some of the nuances:[432]



First thing to note is that a lot of the conclusions we might draw from the expiratory time constant will be known from other realms of clinical practice - history, physical, current treatments, etc. It's also worth noting that the specific issue we might infer from an acutely abnormal expiratory time constant may have already been assessed elsewhere.[433] And then keep in mind that doing the actions listed here won't necessarily fix the underlying pathology - no amount of ventilator manipulation will fix pulmonary fibrosis and, likewise, pharmacologic diuresis is likely needed in combination with vent optimization for the patient with significant pulmonary edema.

Last thing to mention is titration of **Positive End-Expiratory Pressure** while using the expiratory time constant as a gauge of whether or not changes are effective. In **Optimization of PEEP** we mentioned that compliance and driving pressure (among many other things) can be used to gauge whether or not a titration in PEEP was effective or not. Some data suggests that the expiratory time constant can be used similarly.[434] That said, if we have a ventilator that measures an expiratory time constant, it almost certainly also shows compliance. So perhaps we stick with that as a primary assessor when titrating PEEP, but keep in mind that the expiratory time constant may also be utilized. Or if we see the values move contrary to one another (i.e.,

[432] Depta & friends, 2025 - This chart/ graphic is similar to one presented in this article on expiratory time constant, but with a handful of modifications/ clarifications
[433] For example, the lung and airway issues were mentioned previously in both **Watching Pressures** and **Acute Deterioration**
[434] Depta & friends, 2022 - Study on this idea by the same lead author that we cited just a moment ago with the graphic

compliance improves, but RCexp gets worse/ higher/ further away from normal) then we ought to look for other explanations (i.e., maybe one of those airway issues).

Blood Gas Analysis

Blood gas analysis is the process of interpreting the measurements of things in a blood sample to better understand what is happening physiologically with a patient at a given moment in time. The measurements focus on pH (i.e., acidity or alkalinity) of the sample, as well as a few of the factors that are contributing to that value and oxygenation. The idea is that the body will do what it can to keep pH within an ideal range and we can evaluate the mechanisms it uses to do so by assessing these measurements. When viewed as a single data point, our interpretation of blood gases allows us to label what is happening with the patient so that we can make adjustments in the treatment plan to facilitate a move back towards baseline.

When plotted over time, the data points can provide insight on how a patient is doing - either improving or deteriorating - throughout the course of an illness and/ or treatment. In a very simplified way, if the numbers are getting closer to normal the patient is getting better (and similarly if the numbers continue to deviate further from normal the patient is getting worse or more sick). This is particularly useful in mechanical ventilation, because we can alter how these values change by adjusting settings on the vent. There are also point of care machines for blood gas analysis that can be used in the field or during transport, so it's a strategy that may be available to some crews. We've touched on this general idea in previous sections, but we'll lay it all out here as a brief overview.[435]

Just to reiterate, the foundation of this whole thing is that the body will do what it can to try and keep pH in a physiologically normal range. If pH gets too low (acidic), the body will compensate by decreasing carbon dioxide and/ or increasing bicarbonate production. On the other hand, when pH is too high (alkalotic) the body does the opposite - increasing carbon dioxide and/ or decreasing bicarbonate production. Carbon dioxide is adjusted primarily through the respiratory system (i.e., it is correlated with **Minute Volume**) and bicarbonate is created by a metabolic process in the kidneys; hence we have a respiratory and a metabolic component to blood gas or acid-base analysis.[436] And then on top of this we also want to consider oxygenation, as inadequate oxygenation to tissues will result in an increase in anaerobic metabolism that then leads to increased acid production. We won't go into the details of all of these physiologic processes here, as our focus is solely on what we need to know in the context of mechanical ventilation.

With that in mind, the first thing to discuss are what specific data points are measured and what they describe. A normal blood gas sample evaluates the following components: pH, partial pressure of oxygen (PO_2), partial pressure of carbon dioxide (PCO_2), bicarbonate (HCO_3), oxygen saturation (SO_2) and base excess (BE).[437] And then we conceptualize what each of these things represents:

pH	acidity or alkalinity
PO_2	efficacy of oxygenation
PCO_2	efficacy of ventilation/ the respiratory component
HCO_3	the metabolic component
SO_2	efficacy of oxygenation
BE	the metabolic component

[435] See **Patient Already on the Vent** for a list of references on blood gas analysis, no need to repeat them all here
[436] Woodruff, 2007 - This article provides a brief overview of these concepts and outlines a systematic approach to acid base analysis
[437] Base excess is sometimes notated as base deficit when negative, but we'll stick with excess for here; the astute observer may also realize that not all of these values are "blood gases" and that's just the way folks refer to the thing in general

It's worth noting that there seems to be a bit of an overlap here in some of the measurements. We have two things that look at oxygenation and two things to look at what we've labeled the metabolic component. The difference between partial pressure of oxygen and oxygen saturation was already discussed back in **Oxygenation**, so let's focus here on bicarbonate (HCO_3) and base excess (BE). We mentioned in **Acidosis** that there is a difference between the bicarbonate from a blood gas and the bicarbonate from a normal plasma sample (in that one is measured, the other is calculated with an assumed PCO_2), base excess is simply the next iteration of finding the most accurate way to describe this metabolic component.[438] For the vast majority of cases, it's just fine to use the HCO_3 from the blood gas, but know that we could see a normal bicarbonate and an abnormal base excess in some cases.[439]

Next is to consider if we are above or below a normal range for each of these components and assess what that means for the patient:

	below range	above range
pH	acidosis	alkalosis
PO_2	hypoxemia	hyperoxia
PCO_2	alkalosis	acidosis
HCO_3	acidosis	alkalosis
SO_2	hypoxemia	hyperoxia
BE	acidosis	alkalosis

Now to quantify this process, it matters what type of blood sample we are working with. Traditionally we perform blood gas analysis using arterial samples, but it can also be done with venous samples. The normal ranges look like this:[440]

	arterial	venous
pH	7.35 - 7.45	7.30 - 7.43
PO_2	80 - 100	19 - 65
PCO_2	35 - 45	38 - 58
HCO_3	22 - 26	22 - 30
SO_2	95 - 100	23 - 93
BE	-2 - 2	-1.9 - 4.5

On this subject while we are here: even though there is much less data on the use of venous blood gases and even though arterial samples are often considered the standard of care, it is just fine to use venous blood gas analysis in a clinical setting. It also may be difficult to acquire an arterial sample en route while a vehicle is in motion if the patient does not have an arterial line in place. And while it is possible to place an arterial line at bedside in a relatively short amount of time, that may not be in the scope of practice for all transport crews.[441] Our general recommendation is to use an established and consistent line for taking blood samples for blood gas analysis. This both ensures consistency in readings and also prevents undue discomfort to the patient. It can be one that is in place on arrival or one we place at bedside; it can be arterial or venous; if venous, central is preferred.[442] Preference would go to an arterial line, but that may not always be possible.

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[438] Langer & friends, 2022 - More on this idea of base excess here

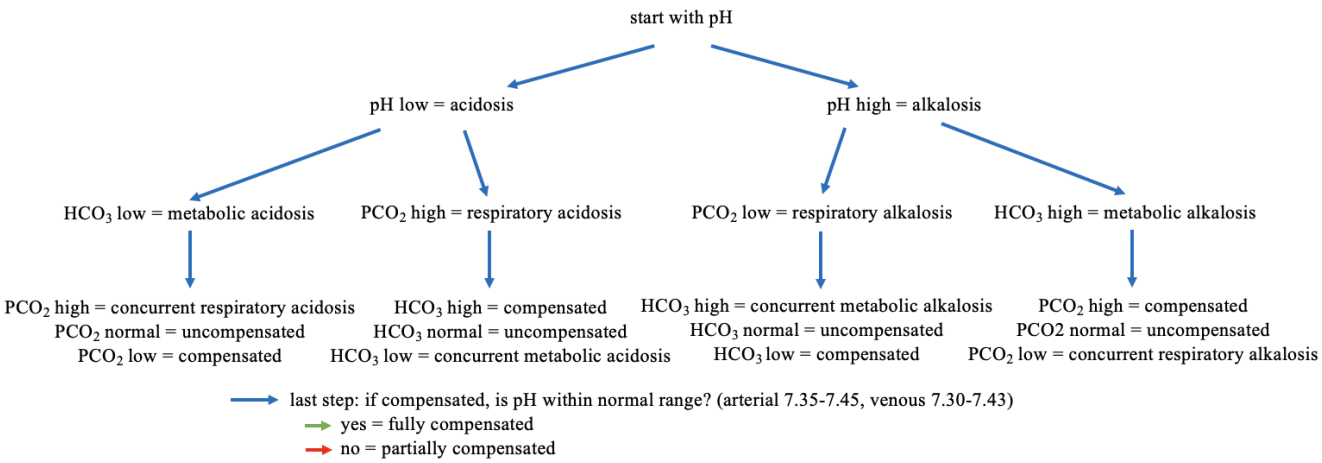
[439] An example of this would be early in a metabolic acidosis caused by the accumulation of acid (such as DKA or some sort of ingestion), the base excess would be abnormal (in the negative direction) and bicarbonate may still be normal

[440] Ress & friends, 2020 - Reference ranges for venous sample taken from this study; just know that different institutions and labs may have slightly different ranges and that's OK

[441] Carley, 2024; Weingart, 2025 - Both of these articles cite that London HEMS has demonstrated the ability to place arterial lines on scene in under two minutes; unable to find a primary source, but send it our way if it's out there!

[442] More on this final point about central versus peripheral samples in just a bit when we discuss **Oxygenation** in a bit more detail

Back to where we were: now we have a list of reference ranges and we know which factors point to which components in the system. Next thing we must do is decide what type of disturbance exists. There are many systems for doing this, but here is one way:



This flowchart can feel a bit misleading at the start if pH is normal or within a normal range. Traditionally when dealing with arterial blood gases, we advise to use physiologic normal (pH 7.40) as the starting point - so with a pH for 7.39, even though it's "normal" we'd call that an acidosis and then continue on down the algorithm. Now we could, theoretically, do the same with venous samples and simply assign the middle point of our normal range as this reference point (i.e., go with 7.37). The idea is that we may have a very slight disorder or one that has already been corrected back to a normal pH.

Once we have an appropriate label on hand, we then need to decide what (if anything) we should do with the vent to address the issue. In general practice, it is often the case that we simply increase **Minute Volume** with states of marked or severe metabolic acidosis to prevent acute decompensation,[443] but there can be more to this process if there is time and headspace available during transport.

For acute respiratory disturbances, it is generally fine to use the vent (i.e., increase minute volume for a respiratory acidosis and decrease it for a respiratory alkalosis) so long as we are appropriately managing the **Three Big Things**. Just keep in mind that there are some chronic conditions that result in atypical or abnormal values for the partial pressure of carbon dioxide and there is no need to try and fix those things in transport. For example, the patient with marked COPD may live with a $PaCO_2$ of 60 - ventilating that down to a "normal" of 40, therefore, isn't necessary. Now the risk of abruptly shifting pH and causing any major adverse effects in the time it takes for a normal transport is low, but that doesn't mean we should simply ventilate everyone to the same goal $PaCO_2$.[444]

[443] As previously discussed in the section on the **Acidosis** vent strategy

[444] Kilburn, 1966 - This older study looked at the consequences of a rapid-onset alkalosis, which is perhaps comparable to overcorrecting a chronic respiratory acidosis in transport; that said, it considers extreme situations and may not indicate that the same would occur in these cases

One other point to make here is that we can do some math to see whether an abnormal PCO_2 is due to an acute or chronic process. Or if we know that a chronic disease process exists, then do the math and find out that our calculated or expected value is different from the measured value, we can infer that an acute process coexists with the chronic problem and then adjust the vent accordingly. This is one of those things that may be best left to reference cards or resources, but here is how an example of one looks:[445]

1-2-3-4-5 Rule	HCO_3	
For every **10mmHg** above/below 40mmHg	ACUTE	CHRONIC
↑pCO_2 (Resp. acidosis)	↑ 1	↑ 4
↓pCO_2 (Resp. alkalosis)	↓ 2	↓ 5

So if we have identified a respiratory acidosis in an arterial sample and see that $PaCO_2$ is 60 (i.e., 2 x 10mmHg above a baseline of 40) and HCO_3 is 28 (i.e., 4 above the normal 24), we know there is some acute component involved. To say it another way, if it were 100% a chronic process, we would expect HCO_3 to be closer to 32 (2 x 4 above the normal 24) - the body would have had much more time to compensate appropriately for the elevated $PaCO_2$. In this case, therefore, it might make sense to adjust vent settings (i.e., increase **Minute Volume**) to a goal $PaCO_2$ of 50, as that's where the $PaCO_2$ ought to be (at least per this quick rule of thumb) based on how far the HCO_3 is from normal.

As another example, let's say we identified a respiratory alkalosis, again on an arterial sample just to keep things easy, and we see a $PaCO_2$ of 25 and an HCO_3 of 17. The $PaCO_2$ is 15 from a normal 40 (i.e., 1.5 x 10mmHg) and the HCO_3 is 7 from a normal 24 (i.e., about 1.5 x 5), which means this is a chronic thing. And therefore we probably don't need to try and fix it with the vent, even though the numbers look weird. Causes for something like this (an isolated and chronic respiratory alkalosis) might include chronic pain, anxiety, some sort of central nervous system dysfunction or prolonged exposure to extreme elevation.

So that's how it works for respiratory disorders - we simply fix the acute components as needed. For metabolic processes there's a similar workflow, but it requires a bit more math. Let's start with the equations for what we would expect $PaCO_2$ to be in either case:

metabolic acidosis: $PaCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$[446]
metabolic alkalosis: $PaCO_2 = (0.7 \times HCO_3^-) + 20 \pm 5$[447]

If the first one looks familiar, that's because we discussed Winter's Formula in **Acidosis** when we mentioned that it may be necessary to increase **Minute Volume** quite a bit to prevent acute decompensation in a state of marked metabolic acidosis. Let's say we have a known metabolic acidosis and an HCO_3 of 15:

$$PaCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$$
$$PaCO_2 = (1.5 \times 15) + 8 \pm 2$$
$$PaCO_2 \approx 31 \pm 2$$

This is to say that if we have a PCO_2 above that value, we should increase minute volume to avoid a worsening of the acidosis.

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[445] Rogers & Cadogan, 2026; Sabatine, 2023 - This shortcut rule comes from this *Life in the Fast Lane* article on acid-base; *Sabatine's Pocket Medicine* also has similar guidance
[446] There is also a shortcut to simply look at the last two digits of the pH; so for a pH of 7.28 aim for a $PaCO_2$ of 28 or lower
[447] Rogers & Cadogan, 2026; Sabatine, 2023 - This formula here is from the *Life in the Fast Lane* article; there are two other slightly different formulas in the Sabatine book, the simplest of which is $PaCO_2 = HCO_3 + 15$

And then for a metabolic alkalosis, let's imagine we have an HCO_3 of 32:

$$PaCO_2 = (0.7 \times HCO_3^-) + 20 \pm 5$$
$$PaCO_2 = (0.7 \times 32) + 20 \pm 5$$
$$PaCO_2 = (22) + 20 \pm 5$$
$$PaCO_2 = 42 \pm 5$$

Which means we could decrease minute volume and allow PCO_2 to rise to 47 or just at the high end of a normal range. Just keep in mind that we still use a weight-based approach to ensuring adequate **Ventilation** and don't deviate far from the necessary 100ml/kg IBW/min.

A quick note on all of the numbers things that we have been doing. These rules and guidelines exist for arterial blood samples only. As far as we can tell there isn't much work that has been done to validate their use with or come up with alternatives for venous blood gases. That said, they can likely be used anyways, as the reference ranges for PCO_2 and HCO_3 between arterial and venous samples are not that drastic and because these sorts of things provide only rough guidance.

Let's recap before we move on. We've gone over all the components of the blood gas and what they mean in isolation. We touched just a bit on the difference between arterial and venous samples. We then worked through how to identify different types of acid-base disorders. For respiratory disorders, we went over how to know if it's acute or chronic and we mentioned that it's really only the acute things we need to focus on with the vent in transport. And then lastly we talked about quantifying where PCO_2 ought to be for metabolic disorders. Next few things to talk about: trending blood gases over time, coordinating PCO_2 with $EtCO_2$, and then just a bit more on oxygenation. After that we'll summarize it all one last time.

Up until now we've discussed blood gas analysis as a single data point in time (i.e., the patient currently has this type of disturbance, therefore we do this thing to try and fix it), but in reality we often trend readings over time. In the hospital setting this may be done once per shift or every few hours, occasionally as often as every hour.[448] Depending on the length of a given transport and patient acuity, we can adjust frequency of analysis accordingly. Regardless of how often we are checking, the idea remains the same: if the patient is trending towards normal, we can likely keep with the current treatment plan. If, however, things look worse, then we may need to adjust to prevent deterioration (as noted above, depending on which type of disorder is present).

It's also worth noting that the vast majority of critical care transport programs do not have access to blood gas analysis in transport. We mentioned towards the start of this section that crews often simply increase **Minute Volume** in (severe metabolic) **Acidosis** and more or less ignore the blood gas otherwise. One reason for this is that real-time data (that would allow us to fine-tune an approach as discussed above) just isn't available. Also because the perceived clinical impact of making minor adjustments in a time-limited transport is not that significant. That said, we can still look at trends in the sending facility's documentation or in printed lab results and get a sense of how things are coming along.

In general, the more recent the blood gas was collected, the better. It is sometimes possible to request a blood gas while at bedside and packaging a patient for transport. This is especially true if the patient has an arterial line in place and if point of care testing is available on the unit (as the effort involved to acquire a sample and run the test is minimal). In the absence of a point of care machine during transport, this would be the best approach - compare the current blood gas to the most recent one and see how things look. If pH is trending back towards an arterial 7.40 (or a venous 7.37) and oxygenation looks OK (more on that in a moment), no significant changes need to be made. If things are looking worse, we can adjust the vent to mitigate the issue and then pass that information on to the receiving facility on arrival.

Now if we do have the ability to analyze a blood gas independently and en route, it may make the most sense to do this initial bedside test ourselves and on our machine. That said, simply having the tool does not imply that we must always use it. There may be cases where the patient looks great, is hemodynamically stable,

[448] To be fair, there are some surgical or operative settings in which blood gases are trended more frequently, but that doesn't happen often once patients make it to the unit or floor

the **Three Big Things** are all just fine, and the hospital's most recent blood gas looked good - in this situation we may decide to proceed with the transfer without running our own blood gas in order to minimize time on scene. Or we may decide to do one en route rather than at bedside. There is no set rule to this sort of thing, just recognize that it isn't always necessary to do simply because we have the capability.

But let's say we do have a really sick patient and the blood gas has been trending in the wrong direction and we make changes to our vent strategy and that we also have the capability to analyze a blood gas en route. In this case it may make sense to recheck a blood gas on the way so that we can make sure we stay ahead of things. Knowing how often to recheck is a bit of a grey area and it depends a bit on what sort of issue we are dealing with. In general pH can change with ventilatory input (i.e., increasing or decreasing **Minute Volume**) on the order of minutes to hours. And this is correlated with the degree of change (i.e., a larger or more drastic change may yield faster results). For oxygenation-related problems, however, it can take much longer.[449] Checking a blood gas at thirty minutes when there is a high level of concern seems like a reasonable starting point. Logistically, this may mean doing one at bedside upon arrival, then again once in flight at cruising altitude - after that we can decide if we need to keep checking frequently or if we can simply do one in a few hours (for long transports) or upon arrival at the receiving facility.[450]

One other advantage of checking a blood gas at bedside is that it allows us to correlate PCO_2 with $EtCO_2$. We mentioned back in **Ventilation** that there is normally a small gap between these two values and that the gap increases with additional **Dead Space**.[451] If we know that there is a real-time, in-the-moment gap of 10mmHg between PCO_2 and $EtCO_2$, this allows us to avoid mis-ventilating the patient. As an example, let's say $PaCO_2$ is perfect at 40, but $EtCO_2$ is 30 (i.e., we have a 10mHg gap between the two). If we only had the $EtCO_2$ to go on, we might be tempted to decrease **Minute Volume** to get an end-tidal closer to normal. But now that we know $PaCO_2$ is fine, we can leave the $EtCO_2$ and minute volume alone in good conscience.

Another use case of this sort of thing is that we can quantify effort to decrease **Dead Space**. So let's say we have a patient ventilated via the **Lung Injury** approach and we are allowing for permissive hypercapnia.[452] We have an $EtCO_2$ of 50 and then measure a PCO_2 of 60 - again, a 10mmHg gap. In an effort to fix this a bit, maybe we remove some amount of mechanical dead space.[453] We allow for some time, then redo the blood gas and now see an $EtCO_2$ of 45 and a PCO_2 53. Slightly smaller gap, slightly better numbers directionally. Next we make some vent changes to recruit alveoli and decrease alveolar dead space. The recheck then shows $EtCO_2$ of 42 and a PCO_2 of 47. Again, slightly smaller gap and slightly better numbers directionally. On the other hand, if we make a change and see a larger gap or numbers going the wrong way, we may want to reconsider and reverse whatever change it was that we made.

The very last thing to discuss here regarding blood gas analysis is **Oxygenation**. So far we've been focused on types of derangements - how to identify and address them in terms of **Ventilation**. But the oxygen side of things is equally important and inadequate oxygenation can coexist with any of these types of derangements. At its most basic level we can use the PO_2 and SO_2 from the blood gas much in the same way as we use the non-invasive SpO_2. Which is to say, if we make a change and these numbers go up, that's great; if they go down, not great. But as will all things, there's a bit more to it than that.

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[449] Refer back to the section on **Hypoxic Pulmonary Vasoconstriction** for a review of this idea

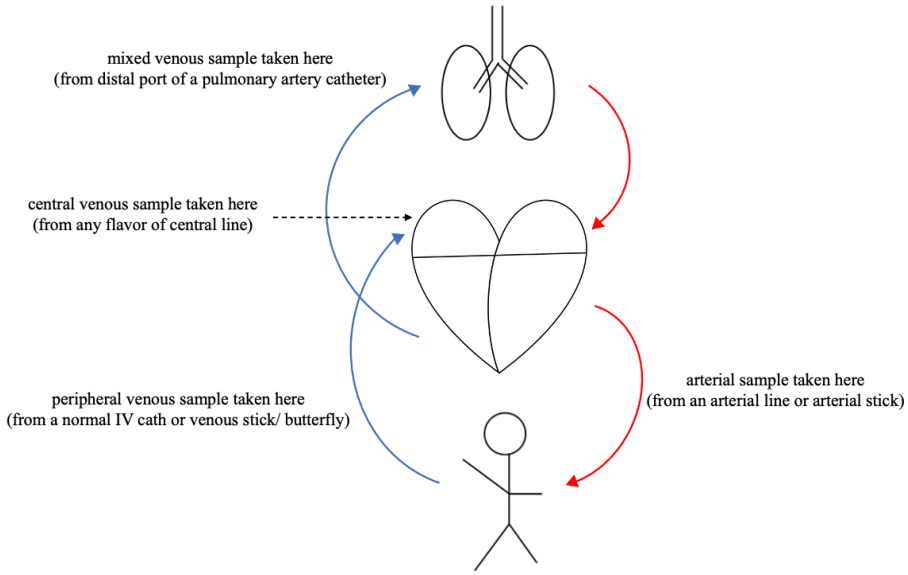
[450] We do recommend repeating a blood gas on arrival or at the conclusion of a transport simply as a learning exercise and to confirm that the changes we made had the intended effect

[451] Yartsev, 2025a - And as cited before, this page describes the idea in detail

[452] Just to review, this would mean a low **Tidal Volume**, high **Positive-End-Expiratory Pressure**, and allowing for an $EtCO_2$ above range

[453] Calculations for this sort of thing is spelled out in the **Appendix** in **Mechanical Dead Space**

Let's start this dive into the nuances of oxygenation via the blood gas with a review of where different types of samples come from:



And then quickly let's peek again at the normal ranges for our measures of oxygenation:

	arterial	venous
PO_2	80 - 100	19 - 65
SO_2	95 - 100	23 - 93

A few things to note here. The normal ranges on the arterial side are much tighter or narrower. This is because there are fewer variables involved - the blood has just recently passed through the lungs to receive its full complement of oxygen and essentially none has yet to be extracted. On the venous side, however, we see that the ranges are highly variable and also much lower. This is because there are more factors involved that take or consume oxygen as it moves through the body. We start with arterial oxygen content (i.e., as blood enters systemic circulation), but then oxygen gets utilized or consumed in the tissues as it passes through. This process is also dependent on cardiac output, as slower movement of blood through tissues allows more time for consumption (i.e., decreased or poor cardiac output results in lower venous oxygenation).[454]

Now it may seem, at first glance, that the venous sample, therefore, is a better measurement because it accounts for more things. To say it another way: in terms of oxygenation, the arterial blood gas only says how well we did at loading oxygen into the blood and onto hemoglobin; the venous sample, on the other hand, does that *plus* provide some insight on consumption/ utilization and cardiac output. The problem with that is that the number of variables involved in the venous sample make it difficult to know if the changes we make in treatment are having the intended effect. Let's say we increase **Positive End-Expiratory Pressure** to try and improve oxygen delivery. If we see an increase in PaO_2 or SaO_2 (arterial values), then we can be fairly certain that the change had its intended effect. But let's say we only have access to PvO_2 or SvO_2; any of the following could be true: improved oxygenation occurred as intended and both PvO_2 and SvO_2 increased; improved oxygenation occurred as intended, but blood return to the heart decreases with increased intrathoracic pressure and the net effect is no change in PvO_2 or SvO_2; or improved oxygenation occurred as intended, but blood return to the heart decreases with increased intrathoracic pressure and the net effect is that both PvO_2 and SvO_2 decrease.

The issue, then, is that the venous sample makes it difficult to know for sure if the change we made was effective or not. This is not to say, however, that it is totally useless, just that we need to be a bit more

[454] Yartsev, 2023f - More on this idea in this discussion from *Deranged Physiology*

discerning when interpreting those venous oxygenation measures. Another consideration with venous samples is where the blood is taken from. While a mixed venous sample from the pulmonary artery would be ideal (since all the venous blood from various return pathways has been mixed in the right ventricle by this point), this is unlikely to be available. Also it may not be necessary. Studies have compared peripheral and central venous measures of oxygenation as well as central venous and mixed venous measures of oxygenation, all with reasonable correlation.[455] Theoretically, local differences in oxygen consumption could skew results, but so long as we utilize the same site for each sample for a given patient, it's probably just fine.

One more point to make on **Oxygenation** and then we'll wrap things up. There are sometimes cases in which oxygenation is quite poor and strategies to fix it are at odds with how we might otherwise choose to manage the ventilator and acid-base derangements. An example of this would be the patient who is septic, has ARDS, and is in a state of metabolic acidosis. In this situation we want to pursue a **Lung Injury** approach to ventilation, but recognize that allowing the patient to become more acidotic could be catastrophic. To reiterate this point - acidosis leads to vasodilation/ loss of vascular tone and decreases the efficacy of many of the typical hemodynamic agents, so allowing an acidosis to worsen can be fatal. That said, inadequate oxygen delivery is also quite bad.

There is, however, the idea that improved oxygenation can lead to vasodilation of the pulmonary vascular beds (reversal of the **Hypoxic Pulmonary Vasoconstriction** effect we have discussed previously**)** which allows for more **Ventilation** or carbon dioxide clearance. So we might actually decide to allow a known acidotic state to get worse in order to improve the situation after some time. We could mitigate the risk to some degree by changing our vasopressor strategy or initiating a bicarbonate infusion.[456] We may also decide to recheck blood gases at more frequent intervals and/ or to have a predetermined hard stop for this strategy (i.e., if pH reaches whatever value, we abandon the experiment). Think of it as one of those "one step back, two steps forward" sort of things.

Another example of this might be a patient with a metabolic alkalosis following significant gastrointestinal fluid loss. Imagine that pH is 7.60, HCO3 is 45 and then we do the math for what an expected PaCO2 would be:

$$PaCO_2 = (0.7 \times HCO_3^-) + 20 \pm 5$$
$$PaCO_2 = (0.7 \times 45) + 20 \pm 5$$
$$PaCO_2 = (31.5) + 20 \pm 5$$
$$PaCO_2 = 51.5 \pm 5$$

And then say that in order to achieve this, we need to drop **Minute Volume** to 75ml/kg IBW/min. At this point, however, we note that PO_2 and SO_2 are lower than expected. This is likely because in an effort to achieve that goal $PaCO_2$, **Mean Airway Pressure** (which is correlated to **Oxygenation**) has decreased. This also puts us at risk of de-recruitment as the lungs are not being filled as much as normal. And then there's the left-shift of the oxygen-hemoglobin dissociation curve to consider here (i.e., oxygen is held a bit more tightly in an alkalotic state). In this case, therefore, we could end up in a hypoxic state trying to prevent an alkalosis from getting worse.

Now in both of these situations just described (the sepsis/ ARDS/ metabolic acidosis combo and the hypoxic metabolic alkalosis example) there are other interventions available beyond the ventilator. We could consider bicarbonate in the first case (as we noted above) or acetazolamide in the second one. We could also involve the receiving ICU team or medical control in that decision-making process if need be, as these may not be things typically initiated by the critical care transport crew. The point of all of this is that there will be times when fixing the acid-base derangement is at odds with ensuring optimal oxygen delivery. And that's OK, it's just something to be aware of.

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[455] Goyal & friends, 2016; El Masry & friends, 2009 - More on this idea in these two papers
[456] More on both of these ideas in **Medications in Mechanical Ventilation**

Time for a final review/ summary on blood gas analysis. We covered how to interpret blood gases, both arterial and venous, in terms of acid-base balance. We talked about when and how to adjust the vent towards specific PCO2 goals in different types of derangements. We explained the difference between snapshots in time and trending at regular intervals, we discussed coordinating PCO_2 with $EtCO_2$, and we considered some of the intricacies of oxygenation and when that might present us with conflicting goals in clinical care. That's a lot of things to consider, so perhaps a checklist would be useful:

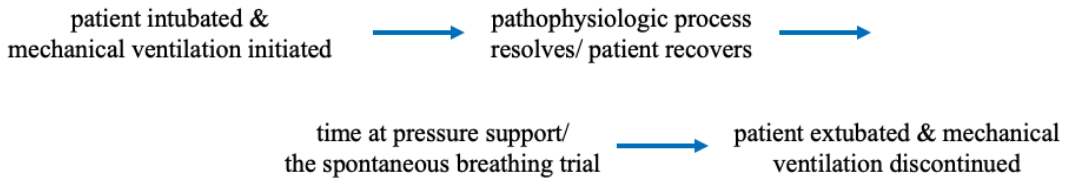
blood gas analysis checklist

- ❑ interpret current or most recent blood gas
  - ❑ consider collection site/ source
  - ❑ consider if HCO_3 and BE match (defer to BE with disagreement)
- ❑ consider direction of PCO_2 change needed
- ❑ if current blood gas available:
  - ❑ quantify PCO_2 goal
  - ❑ assess PCO_2 to $EtCO_2$ gap
- ❑ consider oxygenation
  - ❑ optimize as needed
  - ❑ avoid excess
  - ❑ seek help if oxygenation strategy is at odds with acid-base strategy
- ❑ implement or adjust vent strategy
- ❑ reassess en route as able

Weaning & Pressure Support

Weaning is the process of transitioning a patient from the mechanical ventilator to a spontaneous breathing process. While this isn't something we routinely do in transport, there is value in understanding how the process works, as we may be required to move patients during the process and also because the actions we take while managing patients can affect (both positively and negatively) the workflow.

And pressure support ventilation (PSV) is a mode of ventilation that is often used to facilitate weaning. This isn't to say that weaning is the sole use-case of pressure support ventilation, but it is quite common that the process of going from intubation to mechanical ventilation to eventual extubation passes through some time spent at pressure support ventilation:



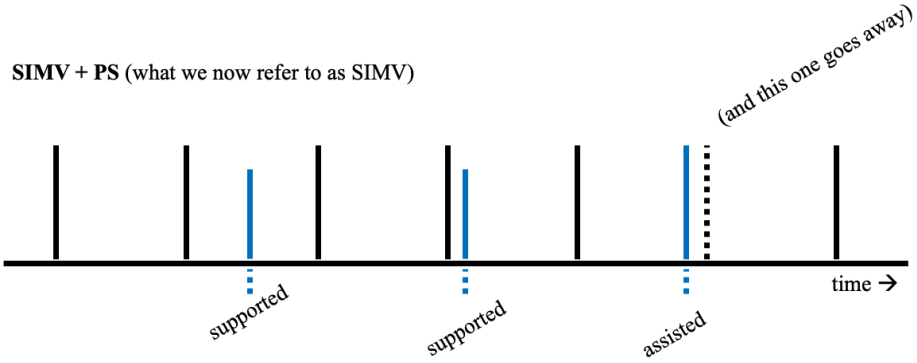
In general, the workflow for getting someone off the ventilator involves a process in which we determine if the initial reason for which the patient was intubated has been resolved and then decide if their current state is sufficient to support ventilation independently (i.e.,without support from the mechanical

ventilator). The process can be quite involved, but suffice it to say that the hope is that we are able to extubate the patient successfully without needing to reintubate them in the near-term.[457]

And then it is worth pointing out one specific step in this process: the spontaneous breathing trial. In this part of the process we place the patient on pressure support as a test, essentially, to see how they do.[458] If all goes well, we can proceed to extubation; if not, we allow for more time and try again some other time or at fixed intervals. One common workflow is to do a trial every shift or every day unless contraindicated. This often involves pausing sedation and analgesia, assessing patient response, then initiating pressure support. With failure along the way (whether due to apnea, agitation, increased work of breathing, hemodynamic instability, etc.), we abandon the trial and then resume a controlled mode of ventilation.

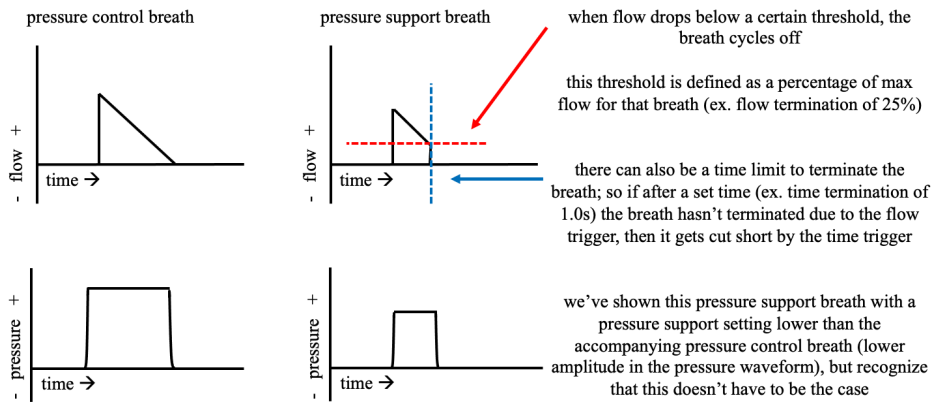
Now along the way we can use the ventilator itself to set the patient up for success with this process. We mentioned in **How is Positive-Pressure Ventilation Different?** that muscle fatigue and/ or weakening can result from mechanical ventilation. There is some thought that pressure support ventilation can mitigate these consequences or at least cause less of them than traditional control modes of ventilation.[459] While more recent data has suggested that it may not be pressure support, in and of itself, that is the best approach, but rather appropriately-titrated levels of support (i.e., patient-specific vent settings), the fact remains that pressure support is a common component of in-hospital mechanical ventilation and weaning.[460]

We initially mentioned the idea of a pressure support breath when we discussed **Synchronized Intermittent Mandatory Ventilation**. At that time we drew out the mode like this:

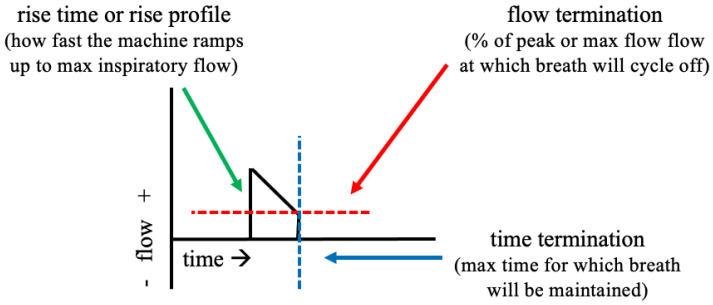


[457] Tobin, 2013 - A seven-step process for weaning is outlined here in Chapter 58 of Tobin's textbook
[458] ICUScenarios.com, 2025 - This podcast/ discussion with Dr. Tobin (whose textbook is cited above) discusses the nuances of the spontaneous breathing trial; specifically, they outline that pressure support is not the only way to do the thing
[459] Brochard & friends, 1989 - This study is often cited to support the use of pressure support in reducing diaphragmatic fatigue and weakness
[460] Schellekens & friends, 2016; Docci & friends, 2024 - The first discusses general strategies for optimizing respiratory muscle function during mechanical ventilation and points out that the data supporting pressure specific alone is scarce; the second goes into the concept of fine-tuning pressure support to best facilitate inherent respiratory function

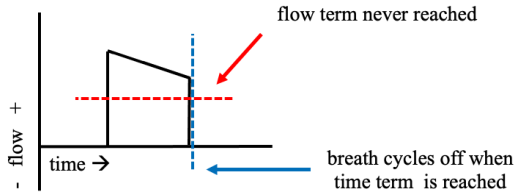
And we said that the smaller breaths (the shorter ones) were *supported* rather than *assisted*. We expanded on that idea in **Types of Breaths** and illustrated the difference between pressure control breaths and pressure support breaths as so:



To expand on this just a bit, we have three parameters we can adjust to manage pressure support breaths. Labels for these vary a bit by manufacturer, but here's the basic idea:



In general, it may help to conceptualize the time termination as a "backup" in the event that the primary flow termination fails. This most commonly happens when there is a leak in the system.[461] And here's how that might look:



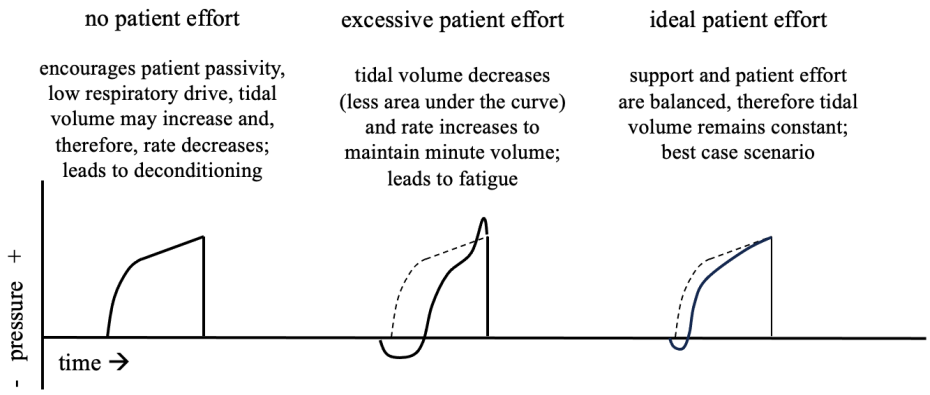
Now when we discuss pressure support as a mode of ventilation, we set the ventilator up so that it only provides supported breaths. To dial this in: in pressure support ventilation, when patient effort meets the **Trigger** threshold, the machine will support that breath by providing a set amount of pressure into the circuit; there is no mechanism for assisted or full breaths, rather all breaths are supported.

The idea here is multi-factorial. First, we encourage patient respiratory effort by requiring that they initiate all breaths. Second, we allow the patient to determine, to some degree, the **Tidal Volume** with each breath (i.e., their intrinsic effort combined with the support provided). And then thirdly, we can then decrease

[461] Tobin, 2013 - As noted here; other reasons could theoretically include inadequate or excessive work of breathing, sensor malfunctions, extraneous movement, etc.

pressure support over time or at set intervals to allow the patient to gradually adapt to less and less support in the hopes to better set them up for eventual discontinuation of mechanical ventilation and extubation - this is where the concept of weaning comes in.

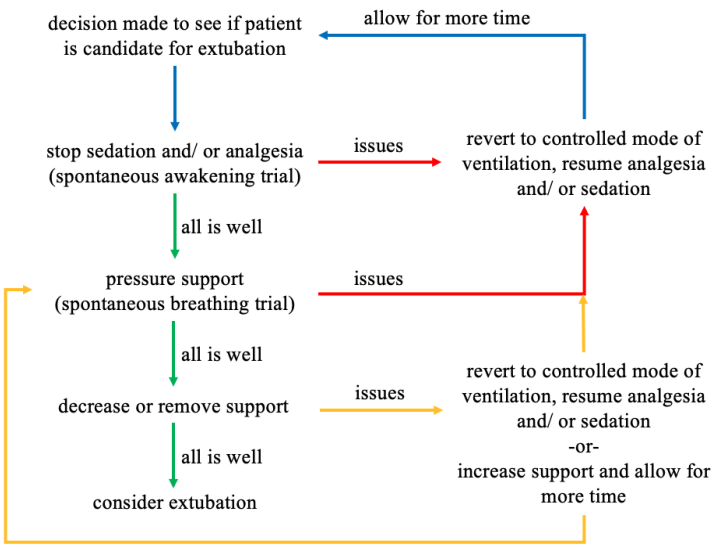
Now before putting this mode to work and implementing a strategy for pressure support ventilation that facilitates eventual discontinuation of mechanical ventilation (i.e., weaning), we need to review a few of the finer points. Most relevant is the idea that the amount of pressure support we provide to each breath is often difficult to pin down and too much or too little can cause problems:[462]



Also there is some concern that pressure support ventilation doesn't mix well with patient sleep and that it can predispose the patient to certain types of **Asynchrony & Dyssynchrony**.[463] While it may be the case that these issues are related to inappropriate pressure support (as noted above), the general trend is worth keeping in mind. And then lastly is the idea that analgesia and/ or sedation can decrease the patient's respiratory drive; this can result in hypoventilation since no backup rate or guaranteed **Minute Volume** exists in pressure support. For this reason analgesic and sedative medications are often discontinued prior to placing a patient on pressure support.

[462] Tobin & friends, 2010; Docci & friends, 2024 - The graphic taken from the first reference, the explanations taken from the second
[463] Tobin, 2013 - More on that in Tobin's textbook on all things mechanical ventilation

All that said, here's what an example workflow might look like:



And then that last step "consider extubation" warrants just a bit of discussion. There are a few ways in which clinicians attempt to predict how well a patient will tolerate extubation in addition to or after the spontaneous breathing trial. Some assessments for this sort of thing include the ability to follow general commands or perform specific tasks (such as lifting the head off the bed) or the ability to cough and/ or mobilize secretions. Also it is quite common to confirm a cuff leak to rule out airway swelling.[464] And then one other thing is the Rapid Shallow Breathing Index - basically a ratio of **Respiratory Rate** to **Tidal Volume**; values less than 105 mean the patient is likely to be AOK, values over 105 mean we should probably let them recover a bit more before extubation.[465]

As for our role as critical care transport folks in this process, we basically have two options when we come across a patient in the midst of this whole process: we can maintain pressure support ventilation or we can put the patient in another mode of ventilation and allow the receiving facility to work back towards pressure support after we drop the patient off. Back when we discussed transitioning a **Patient Already on the Vent** from the hospital machine to the transport machine, we mentioned that it often makes the most sense to simply carry settings over and match what the hospital was doing for the patient. Which would lead us to believe that simply matching pressure support settings is the way to go. This may be OK if the patient doesn't require much in terms of sedation and/ or analgesia, but may not be appropriate if we need those things on the way.

If we do decide to put the patient into a normal mode of ventilation during transport (i.e., one that does guarantee a minimum minute volume), that's totally fine and it allows us to address **Comfort** using medications without the extra concern we'd have if we had stayed in pressure support. And while it might seem intuitive to approximate pressure support settings in **Synchronized Intermittent Mandatory Ventilation** with a low **Respiratory Rate** (so that most breaths are given via pressure support), this may predispose the patient to **Asynchrony & Dyssynchrony**.

Let's summarize. Weaning is the general process by which we transition a patient from mechanical ventilatory support to spontaneous ventilation. This often involves decreasing levels of support via pressure support. Pressure support (the mode) is a mode of ventilation in which all breaths are supported (in contrast to assisted) with a set amount of pressure support (the parameter). Key concepts for the transport crew in pressure support ventilation are ensuring an appropriate level of support and maintaining clinical momentum towards the goal of extubation.

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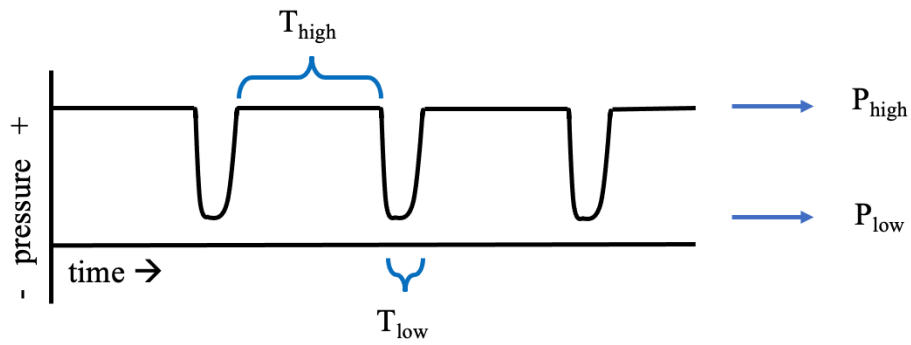
[464] Alrahamaneh, 2020 - Short video on the cuff leak test; also this video is part of a much larger series on mechanical ventilation in general

[465] MDCalc, 2026 - Calculator tool for this guy; also click "evidence" for details on development and validity

Airway Pressure Release Ventilation

Airway Pressure Release Ventilation (APRV) is a mode of ventilation that can be conceptualized as **Pressure Control** with an inverse **I:E Ratio**.[466] Another way to think of it would be a prolonged inspiratory hold or **Recruitment Maneuver** with occasional releases or "sighs" to blow off carbon dioxide. This is how it looks when drawn out:



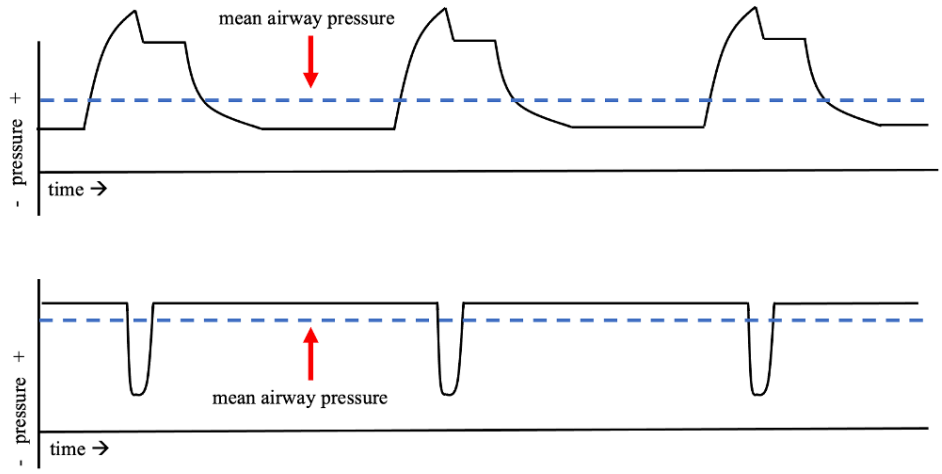
In this illustration we have a pressure-time waveform that shows changes in pressure on the y-axis and time along the x-axis.[467] APRV essentially maintains a pressure over time, as seen by the top line across the graphic where one section is labeled T_{high} and pressure is noted as P_{high}. Interspersed along that continuum of maintained pressure are periodic releases of pressure; these are seen as the downward dips in the waveform, one of which is labeled with the term T_{low} and the correlating pressure is noted as P_{low}.

In APRV we use the ventilator to maintain this high pressure (P_{high}) for long periods of time (described as T_{high}) in an effort to maintain oxygenation. We then allow the pressure to drop to a low point (referred to as P_{low}) at regular intervals (and for a duration of time noted as T_{low}) to facilitate ventilation.

[466] Daoud & friends, 2012 - It is more often described as Bi-Level or BiPAP with an inverted I:E, but that is a concept not discussed in this manual; that tidbit was taken from this overview of APRV

[467] To review this concept in general, look back to **Vent Graphics** and **Waveforms**

The benefit of such an approach is a marked increase in **Mean Airway Pressure** as compared to traditional ventilation. This next graphic is a depiction of mean airway pressure in **Volume Control** ventilation on the top and a comparable graphic depiction of the same concept in APRV. While these two graphics are not to scale, the takeaway is that APRV allows us to maintain an increased mean airway pressure. And we already know that **Oxygenation** is a function of mean airway pressure.[468]



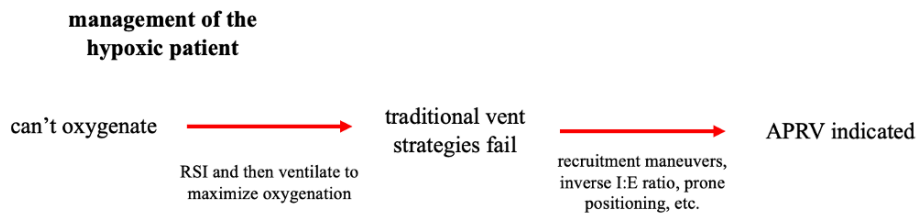
So to summarize before we move on: APRV improves oxygenation via increased mean airway pressure. And increased mean airway pressure leads to improved alveolar recruitment, which further augments oxygenation. Another benefit, and this is key, is that this improved alveolar recruitment facilitates **Ventilation** across the respiratory cycle, much in the same way as **Positive End-Expiratory Pressure**, but to a greater extent. In fact, we may even see improvement in PaCO2 without a corresponding increase in **Minute Volume** as is normally expected in traditional modes of ventilation.

All that said, no vent strategy is without risk and there are some concerns specific to APRV. First is that increased airway pressures can compromise cardiac output even more so than normal positive-pressure ventilation.[469] For that reason, it should be used cautiously in patients with shock. Second is that APRV may make it difficult for a patient to exhale completely. This is of concern for patients who already have difficulty exhaling fully or who otherwise may be prone to AutoPEEP. And third is that APRV may predispose a patient to hypercapnia – we'll address this concept specifically in just a moment, as it may be just fine. One other population to keep in mind are those with chest trauma. The sustained high pressures of APRV can cause a pneumothorax to tension very quickly. While this is a concern with all modes of ventilation, it is especially relevant with APRV.

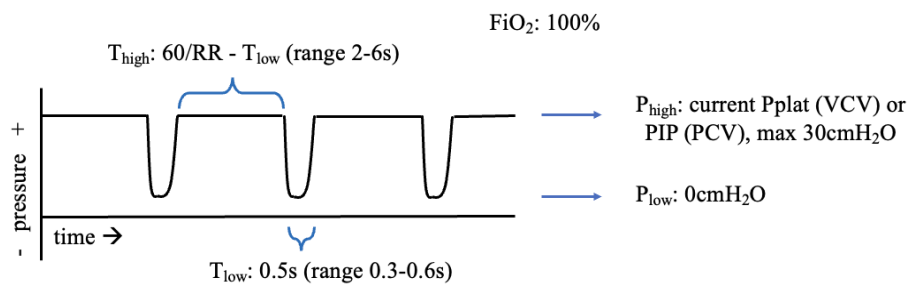
[468] One other thing to note here is that this sustained level of increased airway pressure facilitates alveolar recruitment, in line with a concept known as "open-lung ventilation" that is often discussed in the management of ARDS (this idea was discussed in **Driving Pressure** and **Recruitment Maneuvers**)

[469] This was initially discussed in **How is Positive-Pressure Ventilation Different?**

In terms of utilization, the most common use case is refractory hypoxemia concurrent with ARDS. It is most common to see APRV used as a second-line intervention when alternative strategies to improve oxygenation are not possible or have not been completely effective. For example, it might be used for a patient with ARDS who is not a candidate for **Prone Ventilation** or concurrently with inhaled nitric oxide.[470] In the transport setting we often utilize APRV when it has already been initiated by a sending facility.[471] That said, if we think about a continuum of interventions, as shown in the graphic here, there may be a place for clinicians in the field to implement APRV independently:



As for initial settings when starting a patient on APRV, we've labeled a sample of those on the graphic we used earlier. This is not to say that this is the only way to start a patient on APRV or that there is consensus on these specific numbers, rather it is simply a safe place to begin. Let's discuss each one in turn:[472]



First off is that it's most likely we'll be using APRV to address oxygenation, so an FiO_2 of 1.0 or 100% may be an appropriate starting point. There is always some concern for pathologic consequences of too much oxygen, but in the short term it's completely OK to start at the top and titrate down once we make progress.[473] For T_{low} ,an OK starting point is 0.5 or half of a second. T_{high} is calculated with that value and the current **Respiratory Rate** or simply 5 seconds.[474] As for pressures, P_{high} can be set at the patient's current **Plateau Pressure** or **Peak Inspiratory Pressure** (depending on whether the patient is in **Volume Control** or **Pressure Control**, respectively), ideally under $30cmH_2O$ in either case. We'll talk in just a moment about how we then go about titrating this number to match patient need. P_{low}, on the other hand, is set at zero.

The first step towards individualizing settings on APRV, whether we've initiated the mode using the settings just mentioned or carrying over settings from a sending facility, is to optimize T_{low}. The idea here is that we keep T_{low} to a short-enough interval that alveolar recruitment isn't lost with release breaths, but also

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[470] Discussed in the next chapter, in the section titled **Others Meds in Mechanical Ventilation**

[471] Swindin & friends, 2020 - More on this concept and its use in the ICU setting in this overview

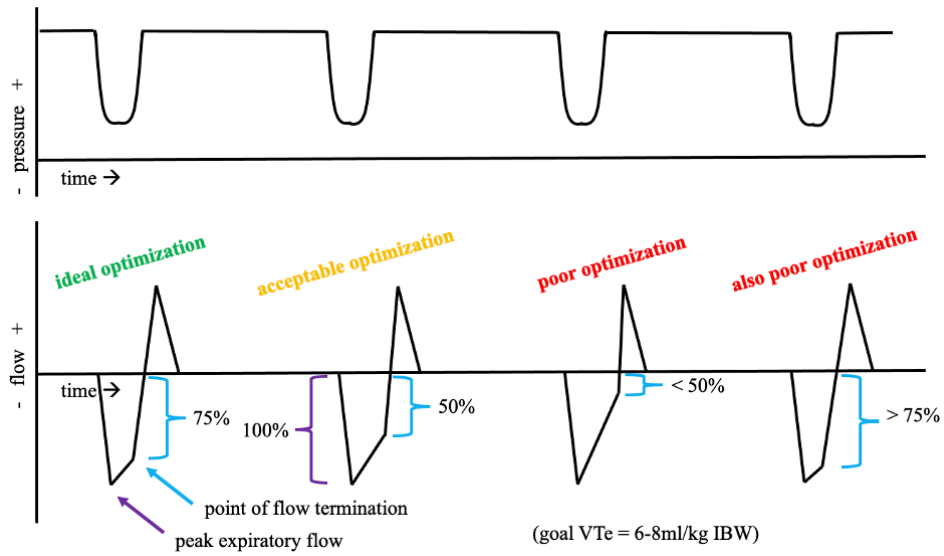
[472] Nieman & friends, 2026 - These starting values are as recommended in this recent paper on APRV, much more detail on it all over there; in severe cases of ARDS or when compliance is very poor, initial settings may need to go beyond these ranges (i.e., P_{high} >30 or T_{high} <3s or T_{low} <0.3s)

[473] This idea was introduced and discussed way back in **Oxygenation**; also refer to our video *Oxygen Haterz* for more on risks of oxygen use in EMS in general

[474] In terms of traditional mechanical ventilation, 0.5s and 5s would yield an inverted I:E ratio of 10:1; seems a bit extreme, but the hope in APRV is that the patient is also breathing spontaneously so this concept of I:E ratio is not an accurate description of what is actually happening – more on that shortly

keep it long enough to allow for exhalation. Let's state both ideas again, just for clarity. If T_{low} is too long and we let pressure get all the way down to zero, we run the risk of alveoli snapping shut or collapsing. Which means we have to work to inflate them all over again and in doing so we increase stress on and damage to the alveolar membranes. And if T_{low} is too short the patient may not exhale fully and we get a buildup of carbon dioxide and a subsequent rise in $PaCO_2$. Which means a worsening respiratory acidosis which may eventually inhibit spontaneous effort to breathe and, thereby, make things even worse.

Our strategy to actually do this T_{low} optimization is two-fold. First, aim for the breath cycling off at 75% of peak flow as a starting point, but know that anything in the range of 50-75% is likely OK. Second is to aim for an exhaled tidal volume of 6-8ml/kg IBW.

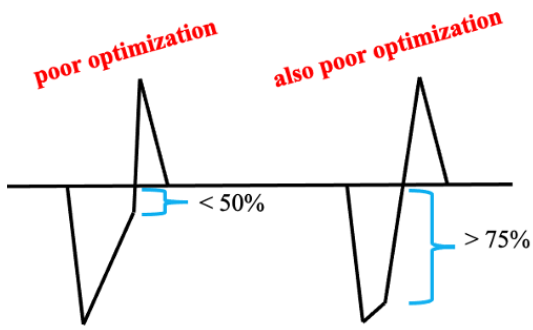


If we look at the waveforms shown in the preceding graphic, ideal is depicted on the left with the breath cycling from passive exhalation or release back towards P_{high} (represented by the steep portion labeled in the blue bracket) when flow has moved towards baseline and is at about 75% of its previous peak. Moving right, the waveform labeled "acceptable optimization" shows this transition occurring when flow has dropped to about half of its previous peak.

Now if, as shown in the third waveform, this transition from release to inhalation happens closer to that zero-flow baseline or at less than 50% of peak expiratory flow, the concern is that de-recruitment of alveoli may occur, and the fix would be to shorten T_{low} or decrease the time spent at P_{low} so that this transition happens sooner. On the other hand, and as shown in the final waveform on the right, if this transition happen too soon or before flow drops to 75% of peak flow, it's quite likely that Vte will be below goal and the fix is to lengthen or increase T_{low} so that more time is spent at P_{low}.

All that said, this balance between adjusting the cycle based on the flow waveform and working towards an ideal exhaled tidal volume can be a bit tricky. If oxygenation is the primary issue, we focus on that and let Vte fall a bit below goal; but if ventilation is the primary issue, we may let this point of flow termination fall below goal. Recognize, however, that even though we differentiate these concepts, better oxygenation facilitates ventilation and vice versa. The distinction just gives us a place to focus on initially.

Just to summarize this concept of optimizing T_{low}. If T_{low} is too long: flow approaches zero, we run the risk of de-recruitment, and oxygenation may suffer. If T_{low} is too short: flow fails to drop to 75% of peak expiratory flow, we run the risk of inadequate ventilation, and Vte may fall below goal. Because of all of this, our first step in troubleshooting with APRV is to take a quick look at the flow waveform and ensure that T_{low} is set correctly.



Once T_{low} has been optimized, we can move on to addressing oxygenation, ventilation, and comfort. We'll start with oxygenation and hypoxia. The quickest fix for the hypoxic patient in any mode of mechanical ventilation is to increase FiO_2.[475] Next step, specific to APRV, is to increase P_{high} in a stepwise fashion up to 30-35cmH_2O.[476] This facilitates recruitment which both increases alveolar surface area and makes it easier for gases to diffuse across the alveolar membrane.[477] And then as an additional action we can prolong the time spent at this higher pressure by increasing or lengthening T_{high}. Increasing P_{high} and/ or T_{high} yields a higher mean airway pressure.

If oxygenation is adequate and we see sustained SpO_2 over our minimum acceptable limit, we can do all of these things in reverse: decrease FiO_2, decrease P_{high}, and decrease T_{high}. That said, we've left "decrease T_{high}" off the list, because once we get to the stage of adequate oxygenation, we generally transition into weaning from APRV and reserve adjusting T_{high} to address ventilation.

So that's oxygenation. Next is ventilation. Evaluating ventilation is less straightforward in APRV than in other **Modes of Ventilation**. In **Assist Control** or **Synchronized Intermittent Mandatory Ventilation** we have two ways to monitor ventilation: a weight-based minute volume goal of 100ml/kg IBW/min (or higher with acidosis) and end-tidal CO_2 with a normal range of 35-45. In APRV that minute volume goal of 100ml/kg IBW/min is still an appropriate measure but know that it may be difficult to achieve, and a certain degree of underventilation or permissive hypercapnia may be tolerable. As for $EtCO_2$ – if we have decent spontaneous effort, $EtCO_2$ waveform and quantitative values may be normal over time. If, however, there is little spontaneous effort, the $EtCO_2$ may only be representative of ventilatory status with those larger release breaths.

With hypoventilation or inadequate tidal volume or high end-tidal CO_2 (which all refer to the same idea) the first consideration is that it is sometimes OK to let ventilation fall below normal via a strategy of permissive hypercapnia. That said, it's still worth trying these other interventions. One thought would be to decrease T_{high} so that release breaths happen more often. The only catch here is that doing so will also cause a decrease in mean airway pressure and could impact oxygenation. Another approach would be to increase P_{high} so that more alveolar volume is recruited. And this approach actually increases mean airway pressure, so it may make sense to both decrease T_{high} and increase P_{high} at the same time to improve ventilation and maintain status quo in terms of oxygenation. And then last option to improve ventilation would be to decrease sedation so that the patient has more spontaneous effort. In fact, spontaneous effort is preferred in APRV and is often targeted with decreased sedation and analgesia.

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[475] Discussed previously in **Oxygenation**
[476] There isn't great consensus on a safe maximum for P_{high} so we left it as a range
[477] Discussed previously in **Alveolar Surface Area**, **Positive End-Expiratory Pressure** & **Oxygenation**

On the other end of the spectrum, if ventilation is well above goal we can either increase T_{high} to allow for fewer release breaths or decrease P_{high} to utilize less lung volume. A few comments here though before we move on. Overventilation in APRV is generally well-tolerated and likely only needs to be addressed at the extreme end of the spectrum. Also recognize that decreasing P_{high} can compromise **Oxygenation**, as this decreases **Mean Airway Pressure**. And then we specifically don't recommend adding sedation to blunt spontaneous effort with hyperventilation, as spontaneous ventilations are recognized to be safest for the patient and the lungs.

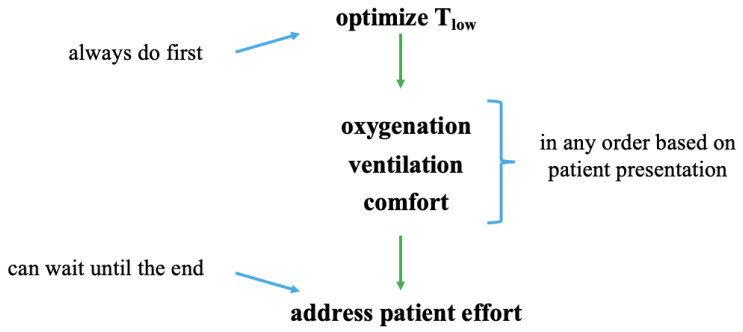
And that leads us into our next subject: patient **Comfort**. If the patient is agitated or otherwise appears to be in pain or discomfort, initial intervention should be analgesia. Sedation may also be appropriate, but we do want to maintain spontaneous respiratory effort as much as possible. And then on the other hand, if the patient is over-sedated and unresponsive or without any spontaneous respiratory effort, the fix would be to wean sedation or allow sedation that has already been given to wear off. The ideal situation, in terms of comfort, would be a patient resting comfortably on the stretcher with no overt signs of agitation or pain and a pressure-time waveform as shown that has spontaneous effort superimposed on the APRV baseline.

The last step in troubleshooting APRV is to address patient effort. This comes after we've optimized T_{low} and addressed ventilation, oxygenation, and comfort. Patient effort is closely related to comfort, but they are distinct concepts. Comfort is addressed with drugs based on patient assessment; patient effort is addressed by manipulating the vent to get the most out of spontaneous efforts.

Now, measuring the total percentage of ventilation done by the patient with spontaneous effort versus that done by the machine with release breaths is a bit difficult, but if we subjectively think effort could be improved and over-sedation has already been ruled out, consider weaning support in an effort to prompt the patient to do more of the work. Specific actions would be to decrease P_{high} and increase T_{high}.

On the other hand, if we think the patient is working too hard and making too much effort, the concern is that he or she may get fatigued and therefore we provide support by increasing P_{high} and decreasing T_{high}. Just keep in mind that this is different than the patient being in pain or being agitated – in those situations we generally treat pharmacologically.

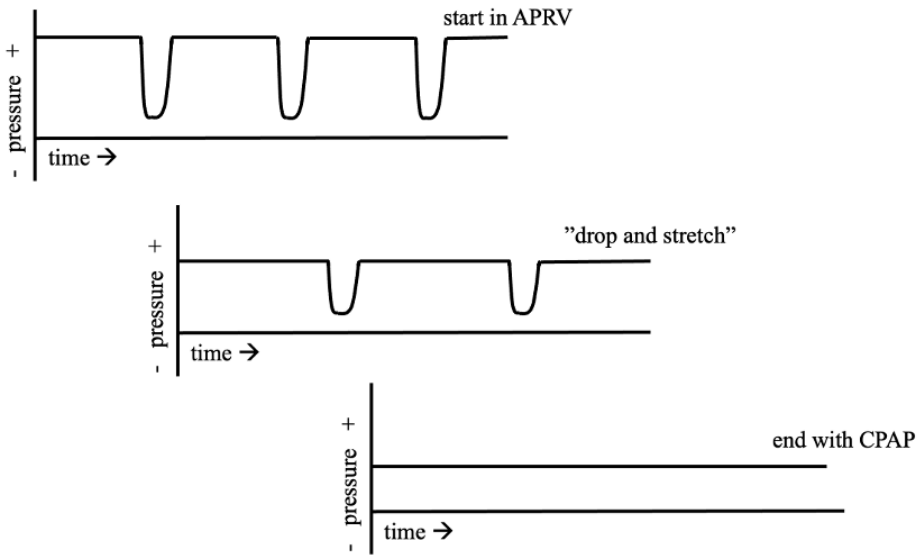
Summarizing things up to this point, here's how we work through troubleshooting and adjusting the details in APRV. We start with optimizing that release breath by adjusting T_{low}. Once that's sorted, we can work through oxygenation, ventilation, and comfort in any order dependent on what we perceive to be most pressing. This may vary from patient to patient and that's OK. And then last thing on our list is to address patient effort and see if we can dial that in to either augment spontaneous effort or provide additional support in order to avoid fatigue.



Two more concepts and then we'll wrap things up. First one is to review how monitored vent parameters might be different in APRV than we are used to with either **Assist Control** or **Synchronized Intermittent Mandatory Ventilation**. We mentioned the first point already: **Minute Volume** may fall below our calculated goal and that is sometimes OK. There are strategies to improve ventilation in APRV, but it may be appropriate to tolerate some degree of permissive hypercapnia or underventilation. Next is exhaled tidal volume: we want to focus on the release breath specifically and are still shooting for a goal of 6-8ml/kg as with other modes of ventilation. Frequency in APRV describes spontaneous effort only, as release breaths are

considered and accounted for by way of other parameters (i.e., T_{high} and T_{low}). As for peak pressure or peak inspiratory pressure, just recognize that we may see values that exceed P_{high} with spontaneous effort, so we do still want to take note of this value. As for plateau pressure, this value is not utilized in APRV. That said, P_{high} is conceptually similar to plateau pressure and may be described as such. We discussed at the very beginning that mean airway pressure is our primary determinant of oxygenation, so this is definitely one to keep an eye on. While there is no established range for mean airway pressure, know that values in APRV will be much higher than we are used to seeing in other modes. And then lastly is end-tidal CO_2 – just remember that it may vary with time and that the most accurate representation of ventilation will likely be on those release breaths.

Very last thing and then we'll wrap it up. Weaning off of APRV is something we likely won't do in transport, but it does help to visualize how this transition from APRV happens over time. At the top of the next graphic we see a pressure-time waveform characteristic of someone on APRV. If all is well and we feel that it is appropriate to decrease mechanical support, we dial down P_{high} and dial up T_{high}. In the context of weaning, we refer to this pair of adjustments as "drop and stretch." Moving from top to bottom, we see that if this sequence of adjustments is repeated over time we wind up with a consistent level of positive pressure or CPAP. The next step would be to conduct a spontaneous breathing trial and assess the patient's readiness for extubation.[478] Again, this isn't something we expect folks will do in transport, rather this is simply to demonstrate next steps in the management of APRV.



Just to review the macro-level steps in APRV, let's do one final summary. Once initial settings are dialed in, the first step is to optimize T_{low}. From there we can address the Three Big Things: oxygenation, ventilation and comfort. And then if time allows, we can focus on patient effort and then work towards decreasing support for eventual transition off of APRV.

[478] Discussed in the previous section, **Weaning & Pressure Support**

Medications in Mechanical Ventilation

This here section is a brief overview of medications that are often used in the management of patients who are being ventilated mechanically. It is no way an exhaustive list nor a substitute for focused references and in-depth study on the subject of pharmacology, rather it is a simple narrative that covers different medications and how they play into the overall game of mechanical ventilation in the prehospital setting.

Agents for Comfort and/ or Synchrony

This idea that we need to keep patients comfortable on the vent has been discussed in a number of places along the way in this book, mostly in the sections on **Comfort** and **Asynchrony & Dyssynchrony**. While there are often non-pharmacologic things we can do to make this happen, sometimes medications are necessary. And quite often these medications are first-line and assumed to be a given. With that in mind, let's review the landscape of different agents and point out a few things along the way.

The first chunk of things that fall into this category are analgesics. These most often include opiates or narcotics such as fentanyl and morphine, but may also include less-potent things such as IV acetaminophen (paracetamol) or IV/IM non-steroidal anti-inflammatory drugs (NSAIDs). NSAIDs of this nature would include things like ketorolac or diclofenac.[479] Now the data on using acetaminophen and/ or NSAIDs in place of more potent agents isn't great,[480] but there is fairly good evidence that concurrent use of them allows for lower/ decreased doses of those other things.[481] Which is potentially of value in terms of weaning[482] and avoiding delirium.[483]

Another option for analgesia that also leads us into the next subject of sedative medications is ketamine.[484] Most folks who work EMS these days are aware of the dose-dependent effects of this medication - analgesia at lower doses, sedation at higher doses, then dissociative doses beyond that. It can be given in push-doses or as an infusion, is generally considered fairly safe in terms of hemodynamic side effects, and even has some bronchodilatory effects which make it a reasonable choice in **Obstruction** (discussed next). For all of these reasons, ketamine is used quite frequently for analgesia and sedation with intubated patients in the out-of-hospital setting. That said, convincing data is still pending and, therefore, we still tend to see other agents used in many hospital settings.[485]

Those other sedative agents (the ones more commonly used in hospital settings, but also often available prehospitally) include things like benzodiazepines (primarily midazolam), propofol and dexmedetomidine. Benzodiazepines are fairly well understood by EMS clinicians, as they are used in many systems and for both sedation and other things (i.e., seizures). One thing to make note of here is that a major concern with these medications is hypotension and that this is more profound in hypovolemic patients (i.e., appropriate resuscitation is key to mitigate this effect).[486] There is also some concern about the effect benzos can have on weaning and their contribution to ICU delirium.[487]

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[479] And less commonly, things like dexketoprofen

[480] Mahshidfar & friends, 2016 - This study showed no decreased need for morphine and no reduction of pain severity when IV paracetamol was used for analgesia with intubated patients

[481] Ma & friends, 2023 - This review shows NSAID use in the ICU does reduce opioid use, but may not impact overall outcomes

[482] See **Weaning & Pressure Support** for a review of this concept

[483] Duprey & friends, 2021 - See here for more on ICU delirium and opioid use

[484] Yartsev, 2023d - See here for all there is to know about ketamine; also links on the right of this page for similar discussions of the other sedative agents discussed next (midazolam, propofol, & dexmedetomidine)

[485] Fuller & friends, 2019 - This review attempted to collate data on ketamine use with intubated patients, it found unconvincing evidence of benefit with ketamine use

[486] Adams & friends, 1985 - Older paper that first investigated this idea using mega-doses of midazolam on dogs

[487] Skrobik, 2012 - This opinion piece is in favor of benzodiazepines for sedation of mechanically ventilated patients, but it does review and address many of the concerns that folks have with this strategy

Next on the list is propofol. Propofol is used quite often in hospital settings for ongoing sedation of mechanically ventilated patients, but is less common in transport.[488] There is a pervasive misconception amongst US-based critical care transport practitioners that propofol is ineffective in transport due to external stimuli (such as noise, vibrations, etc. - all the "stressors of flight"). That said, this is not supported by data and there are good reasons to consider using propofol in transport. One is continuity of care: if a patient is on propofol before transport and will likely be on it again after, it may make sense to limit the number of variables involved in the care of the patient. Another is that propofol has a short half-life and is easy to titrate off as needed if patient condition changes.[489] Less common, and arguably more obscure, considerations in favor of propofol include prevention of nausea, availability of push-doses[490] as needed, anti-seizure properties and amnesic effects.

A few notes, however, on the use of propofol in transport. It has minimal analgesic effects and is almost exclusively a sedative agent, so consider concurrent pain management if needed. Also recognize that hypotension is a known adverse effect and that it is more likely in the setting of inadequate resuscitation. There is also concern for Propofol Infusion Syndrome with long-term use and increased risk of infection/ contamination in the line due to its nature as a lipid emulsion - neither of which is particularly relevant to transport, but worth being aware of. And then last thing to mention is that propofol can cause respiratory depression; while this is more relevant in the hospital setting when used for procedural sedation, it may be relevant with patients being ventilated in transport.

Last agent to mention is dexmedetomidine, also known as Precedex. This one is not very common in transport at all, but within the realm of possibility for crews to encounter. Dexmedetomidine is unique in that it causes sedation via a different pathway than most sedatives (α-2 versus GABA).[491] This preserves respiratory function and often allows the patient to remain awake to some degree and, therefore, able to cooperate and interact with healthcare providers. As far as side effects go, it can cause bradycardia as well as both hyper- and hypotension. It can be used along with other agents or in isolation, may involve an initial loading dose, and does not cause amnesia as is common with some other sedatives.

Moving on from sedatives, the next general class to consider are paralytics.[492] We've mentioned paralytics a handful of times before, but always with the caveat that they should be reserved for extreme circumstances - issues of safety and ventilator strategies that are known to cause extreme discomfort. There are a number of agents available, but in broad strokes we most often use non-depolarizing and longer-acting agents. Among these are rocuronium, vecuronium, and cisatracurium (also known as Nimbex); and of these, cisatracurium has been studied the most in prolonged paralysis of ventilated patients. Paralytics for continuous paralysis are generally given via an infusion, but this can also be achieved with push-doses at regular intervals.

Proposed benefits of paralysis include decreased incidence of **Asynchrony & Dyssynchrony** as well as decreased metabolic activity (i.e., decreased oxygen consumption and decreased carbon dioxide production). There's also arguments to be made for using paralytics in hyperthermia to avoid heat generation in skeletal muscle and in therapeutic hypothermia to avoid shivering (when refractory to all the other things). Another use case would be to facilitate performance of a procedure (i.e., endotracheal tube exchange or, less likely, chest tube placement[493]) - in these cases it could even make sense to use a short-acting agent such as succinylcholine and there would be no need for a continuous infusion (i.e., just give a push to facilitate the thing).

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[488] Moy & friends, 2021 - The AIR SED study identifies this discrepancy (i.e., infrequent use of propofol in transport; at least in the US where the study was conducted), in spite of the fact that it "...has been almost uniformly associated, in over 2 decades of research, with improved outcomes when compared with benzodiazepines"

[489] Platnick & Chatterjee, 2023 - This and the following tidbits (here in this paragraph and the next) are mentioned in this brief overview of propofol

[490] Just be aware that this sort of thing is explicitly prohibited in some places for certain professions, always act within an established scope of practice!

[491] Gerlach & Murphy, 2011 - Overview of dexmedetomidine use in the ICU setting; all the things in this paragraph are discussed here

[492] Farkas, 2025b - Further reading on the use of paralytics in mechanical ventilation

[493] As a sidenote on this point: we mentioned before in **Acute Deterioration** that it probably makes sense to place a chest tube prior to positive-pressure ventilation if possible, but that may not always be possible

One other thing to mention in this discussion of paralytics are reversal agents. These are not routinely carried by transport crews or used in transport (at least in the US), but it is worth knowing that they exist. The two agents most likely encountered are sugammadex[494] and neostigmine[495], but there are others as well. Sugammadex works against rocuronium and vecuronium (not cisatracurium); neostigmine works against all three of these non-depolarizing agents.[496] Just know that these reversal agents are most commonly used in anesthesia and the use case for these things to mitigate issues related to comfort is basically nonexistent. The rationale for reviewing these is that it is within the realm of possibility that a paralytic is reversed by anesthesia while a transport crew is at bedside; knowing to expect reversal of paralysis and to anticipate the need for other agents to address comfort is key.

That's just a brief overview of analgesics, sedatives and paralytics - the medications we might use to ensure patient **Comfort** during mechanical ventilation. This list is not exhaustive and there may be other things out there, especially for those who work internationally or in austere settings. And then don't forget that drugs are just one component of the comfort strategy - also consider adjusting vent settings based on findings seen in **Vent Graphics**.

Medications for Obstruction

When we introduced the idea of **Specific Vent Strategies**, the first one we discussed was **Obstruction**. We mentioned then that vent strategy is just one part of management and is focused on avoiding decompensation or accommodating the pathologic process. Pharmacologic intervention, on the other hand, is what will fix the problem at its root cause. We'll discuss these all in turn, but medications for obstruction include bronchodilators (albuterol, terbutaline, epinephrine, magnesium sulfate and ketamine), anticholinergics (ipratropium) and steroids (dexamethasone, methylprednisolone). Just note that this list of medications is not exhaustive and that some of these agents have other functions in addition to the groups we've allocated them to.

Starting with the largest group here, we have the bronchodilators. As mentioned earlier on, obstruction is an issue where the airways become narrowed due to constriction and/ or mucus buildup. Medications that cause the bronchioles to dilate or open up are, therefore, the mainstay of treatment. Most common is the beta-agonist albuterol (also known as salbutamol in some places), which is very common and likely already a known agent for most anyone reading this. One thing to note is that albuterol exists as a combination of two stereoisomers, a left-hand version which is thought to cause the bronchodilatory effect and a right-hand version which is believed to cause some of the untoward side effects (such as tachycardia, even bronchoconstriction!).[497] It is possible to get preparations of that left-hand isomer only; it is known as levalbuterol (or Xopenex in the US). And regardless of which inhaled beta-agonist is available, everyone should know how to administer these agents via **In-line Nebulization**.

Another beta-agonist that is commonly available to transport providers is terbutaline. In critical care transport we tend to lump terbutaline into the category of things used for obstetrical emergencies as it is often used as a tocolytic, however that's actually an off-label use for the drug and its primary use (at least in the US per the FDA) is as a bronchodilator.[498] It is commonly given via SQ or IM injection,[499] but also can be given IV or nebulized like other beta-agonists.[500] And then it is worth noting that terbutaline has comparable efficacy to epinephrine in some studies.[501]

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[494] Faulk, 2023b - More on Sugammadex here
[495] Faulk, 2023a - And more on Neostigmine here
[496] And neither are effective against succinylcholine, but that's OK because it is short-acting
[497] Ameredes & Calhoun, 2009 - Further reading on levalbuterol; and the main point here is that the left-handed isomer (levalbuterol) binds more strongly to the beta-2 receptor sites and that most of the adverse effects are caused by action of the right-handed isomer
[498] DailyMed, 2024 - Further information on terbutaline approval and warnings in the US
[499] Johansen, 1974 - Not much research into the comparison of SQ vs. IM terbutaline since this older paper…
[500] Pierce & friends, 1981 - Another older paper looking at IV and nebulized administration of terbutaline; says both are effective, but IV causes more systemic effects
[501] Alsabri & friends, 2025 - This systematic review looked at the efficacy of terbutaline and epinephrine for the treatment of pediatric asthma

Epinephrine is another bronchodilator that most prehospital providers will already be familiar with. With severe bronchoconstriction we most often give an IM/ SQ injection,[502] but may also initiate a continuous IV infusion. No need to review all that here, just be sure to know these doses and workflows in and out or have ready access to reference materials if needed emergently.[503] And it is true that epinephrine is often categorized as other sorts of things, but in reference to bronchospasm the primary property we want is the bronchodilatory effect. Also recall that bronchospasm can occur simultaneously with or as part of anaphylaxis - in these cases epinephrine is preferred for reasons beyond its bronchodilatory effects.

Other agents in this category of medications that have bronchodilatory effects, but are more commonly used for and labeled as other things (i.e., not typically categorized as bronchodilators) include magnesium sulfate and ketamine. Evidence surrounding the use of magnesium in asthma is a bit of a mixed bag, but it does seem to have some value and is a reasonable adjunct in severe cases after other agents have been trialed.[504] It is most commonly given as a slow IV bolus over at least fifteen minutes, occasionally as a continuous infusion, and rarely as an inhaled agent;[505] it should not be given as a rapid IV push due to the risks of hypotension and respiratory depression.[506]

Last agent in our discussion of bronchodilators is ketamine.[507] Now in our setting, bronchodilation is mostly just a convenient side effect of ketamine administration; we don't typically give ketamine for that reason alone. That said, it does have value in refractory cases due the fact that its less-potent bronchodilatory effects occur via mechanisms (yes, there are multiple mechanisms involved) other than direct action on beta receptors.[508] So in the case of beta receptor desensitization or downregulation in the setting of prolonged use of beta-agonists, ketamine can come to the rescue. This is also why it is often preferred for analgesia and/ or sedation in patients with issues related to bronchoconstriction. And it can be given via IV bolus, IV infusion or nebulization.[509]

Moving on from bronchodilators are the anticholinergics, of which the primary one used in emergency settings is ipratropium (Atrovent). Anticholinergics work to mitigate secretions in the airway, induce smooth muscle relaxation and inhibit some parasympathetic response.[510] Regarding ipratropium specifically, it lasts about six hours, therefore is often given as a single dose in the treatment of bronchoconstriction.[511] That said, there is some data suggesting that repeated doses of ipratropium yield better results in some cases.[512] The takeaway here is that in refractory cases or in prolonged bronchospasm, it may be necessary to give ipratropium following the initial dose.

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[502] Tanimoto & friends, 2023 - Brief discussion on IM vs. SQ epinephrine
[503] Consider using the Field Reference Guides or (H)EMS Drug Guide found at rykerrmedical.com
[504] Rovsing & friends, 2023; Ni & friends, 2022 - A systematic review on mag sulfate in asthma; another one on its use in COPD
[505] Knightly & friends, 2017; Rovsing & friends, 2023 - This 2017 Cochrane Review showed minimal benefit from inhaled mag sulfate; the 2023 systematic review discusses this further
[506] Patel, 2025 - More on the adverse effects of mag sulfate here
[507] Frentzel, 2021; Binsaeedu & friends, 2023 - Two general reviews on the use of ketamine in asthma, the first in general and the second specific to peds
[508] Ekong & friends, 2025 - This idea and the various mechanisms are discussed in this paper that just so happens to look at both ketamine (nebulized) and mag sulfate for the treatment of asthma
[509] Hemmingsen & friends, 1994 - This older study looked at the effects of an IV bolus dose of ketamine on bronchospasm in mechanically ventilated patients
[510] Barbour-Taylor & friends, 2024 - More on pharm stuff in general in this free textbook from OpenStax
[511] Rodrigo[2], 2002 - This discussion of anticholinergics in asthma dives into lots of the nitty gritty detail, including why we don't just use atropine anymore
[512] Ducharme, 2013 - This tidbit is buried in the Cochrane Review and is specific to pediatric asthma

After that we have the steroids, specifically the corticosteroids, and then even more specifically the glucocorticoids. It gets complicated and doesn't much matter, but here's a quick overview for those who do care:[513]

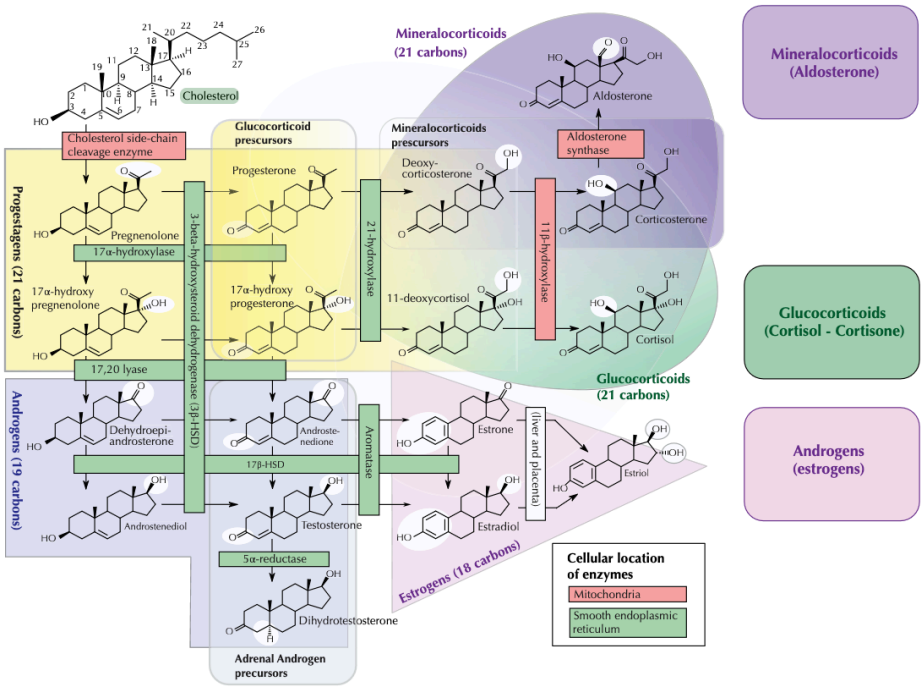


Figure 22-13 Adrenal steroidogenesis. Note how all the steroid hormones are derived from cholesterol. *Source:* https://commons.wikimedia.org/wiki/File:Steroidogenesis.svg. Creative Commons Attribution-Share Alike 3.0 Unported license.

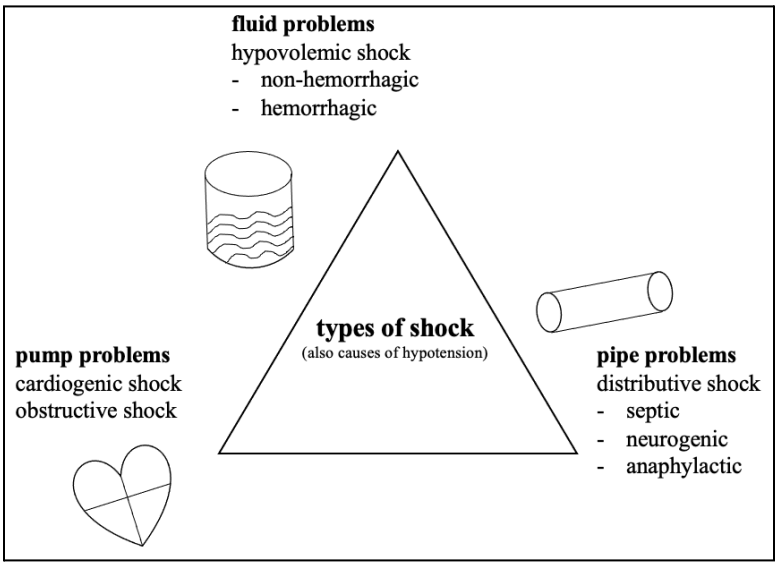
Things like hydrocortisone, prednisolone, methylprednisolone and dexamethasone are often used in transport via single IV bolus doses.[514] In other settings some of these and other glucocorticoids are given PO or by inhalation. On that note, we will point out that there is data to support the oral administration of the IV preparation of dexamethasone (i.e., just draw up in a syringe and give by mouth)[515] - this isn't super relevant for most patients who are being mechanically ventilated, but it could potentially be useful in less severe cases or may have been done prior to intubation.

Other agents for the treatment of asthma that we don't generally see in transport would be things like theophylline/ aminophylline, Heliox or inhaled anesthetics (Sevoflurane or Isoflurane). We won't spend any time discussing those, but just know that in very rare cases you may come across patients who have been given these agents during their course of treatment.

[513] Schifter & friends, 2021 - Textbook chapter on endocrine disorders that explains the origins and functions of different steroid molecules
[514] And these are listed in order of least potent to most potent
[515] Volk & friends, 2018 - More on this concept here

Therapeutics for Hypotension

Hypotension is quite common with mechanical ventilation,[516] so it makes sense to review what we can do pharmacologically to fix it. And it helps to consider what is the underlying cause of a patient's hypotension when deciding how to go about setting things right. There is a simple concept often used in EMS to differentiate the different causes of shock (and thus hypotension, even though these concepts aren't necessarily interchangeable):



Starting with the non-hemorrhagic, hypovolemic option we have IV fluids. Lactated Ringer's (or a comparable "balanced solution") is generally preferred over normal saline, and these crystalloids are often preferred to colloids (such as albumin or hydroxyethyl starch) on the basis of cost even though there is some data to suggest colloids may have benefit (i.e., achieve desired outcomes with lower overall volume given, maybe even improved outcomes overall in some cases).[517] Specific to vent management, there is concern that excessive IV fluid administration can lead to more time spent on the vent and worse outcomes.[518]

One way to mitigate overuse of IV fluids is by evaluating fluid responsiveness.[519] This applies to all patients who get fluids, but with mechanically ventilated patients there are some specific techniques that can be used. For example, nearly all ventilated patients in transport are monitored with continuous waveform capnography; the passive leg test can be done with a significant change in EtCO2 indicating that the patient is, in fact, fluid responsive. Another example would be the use of an end-expiratory occlusion if there is an invasive line in place that allows real-time monitoring of cardiac output.[520] There are many techniques, but the takeaway is that IV fluids are not benign and, therefore, their need should be evaluated prior to administration.

Moving on to hemorrhagic hypovolemia we have blood products to discuss. The main thing to be aware of is that there is a complication called transfusion-associated acute lung injury (TRALI), which is basically

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[516] As we discussed in **How is Positive-Pressure Ventilation Different?**, mechanical ventilation can decrease preload to the heart and predispose the patient to hypotension

[517] Prescott & friends, 2026; Hemming & friends, 2017 - This first paper from the Surviving Sepsis Campaign discusses IV fluids in depth, not just in sepsis, they note that normal saline may be preferred in traumatic brain injury; the second is a subgroup analysis of another study showing benefit of colloids over crystalloids

[518] Mourik & friends, 2019 - This paper looked specifically at ARDS cases, so it's not 100% clear if this is generalizable to all vented patients

[519] Mark, 2021 - This quick reference from ICU One Pager reviews these and many other techniques for evaluating fluid responsiveness

[520] Not common in transport these days, but this is likely to become more common in the coming years

Lung Injury caused by blood product administration.[521] Mechanical ventilation (especially with high **Peak Inspiratory Pressure**) and the use of whole blood or plasma (versus platelets or PRBCs) increases the risk. The pathophysiology of how it all works is complicated and involves multiple pathways, but there are some ways we can mitigate the risk of TRALI: use blood only when indicated, avoid excessive pressures on the vent and avoid concurrent fluid overload (i.e., excessive IV fluids, see above). One last thing: rapid administration of blood is not an independent risk factor for TRALI, but rapid administration can be associated with transfusion-associated circulatory overload (TACO) and TACO includes an element of pulmonary edema that can be detrimental to ventilated patients.[522] So consider giving blood products at a reasonable, slower rate when possible and prudent to do so.

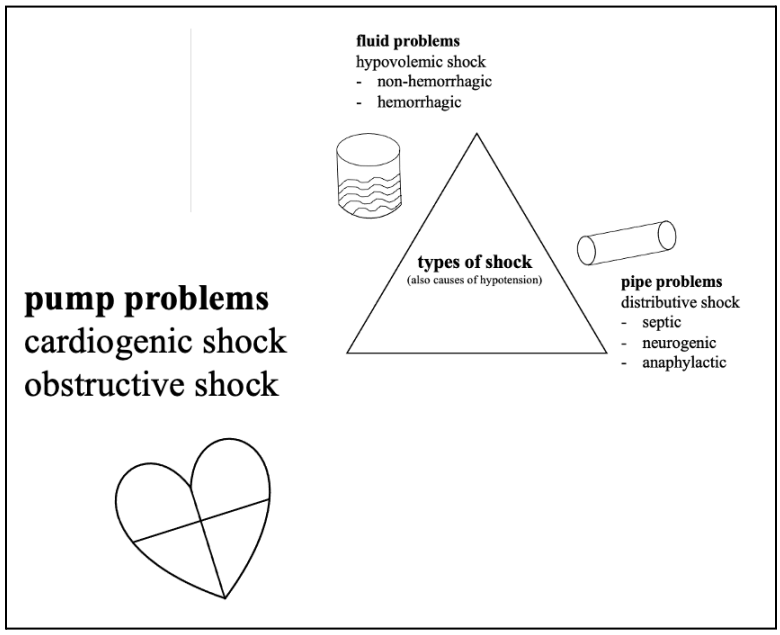
Two other agents that are often given concurrently with blood products in hemorrhagic shock are tranexamic acid (TXA) and calcium salts (generally calcium gluconate, sometimes calcium chloride). So far as we can gather, there are no specific concerns for either of these in the context of mechanical ventilation in transport other than the fact that hypocalcemia can cause diaphragmatic weakness in ventilated patients.[523]

On to hypotension related to distributive shock states: after IV fluids and treatment of the underlying condition (i.e., antibiotics in sepsis or epinephrine and removal/ discontinuation of the source in anaphylaxis), the next category of medications are the vasopressors. In general there is a ton of information out there on the use of vasopressors in critical care medicine, emergency medicine and also transport medicine, so we'll just mention a few pointed facts here that may help specifically in the management of ventilated patients:[524]

- Epinephrine is generally preferred with **Obstruction**/ bronchoconstriction for its beta-agonist effects; dopamine may also result in some meaningful bronchodilation if another agent is needed[525]
- Vasopressin is often cited as the preferred agent in profound **Acidosis**, but that claim has recently been questioned by some[526]
- Norepinephrine is often the preferred agent in **Lung Injury** and when in doubt in general
- Vasopressin and/ or Norepinephrine do not cause vasoconstriction of the pulmonary vascular bed and may be preferred with concern for pulmonary hypertension or states of right-sided heart failure

[521] Hu & friends, 2021 - Review of risk factors related to TRALI
[522] Semple & friends, 2019 - Discussion of both TRALI and TACO
[523] McCool & Tzelepis, 2021 - In fact, all kind of electrolyte abnormalities can impact diaphragm function
[524] Farkas, 2024c - And an excellent place to start would be the IBCC Chapter on "Shock and vasoactive medications"
[525] Fodor & friends, 2019 - Just be aware that the combo of Epi + Dopamine can cause issues with dysrhythmias/ ectopy, so be careful here; also this finding has not yet been validated in humans, so far as we can tell
[526] Bauer & friends, 2022 - More on that idea here

And then we have agents related to pump problems, as depicted in the graphic we started with:



Obstructive shock is the most straightforward, so let's start there. Most of these things are not resolved with medications, rather with interventions or procedures as outlined below:

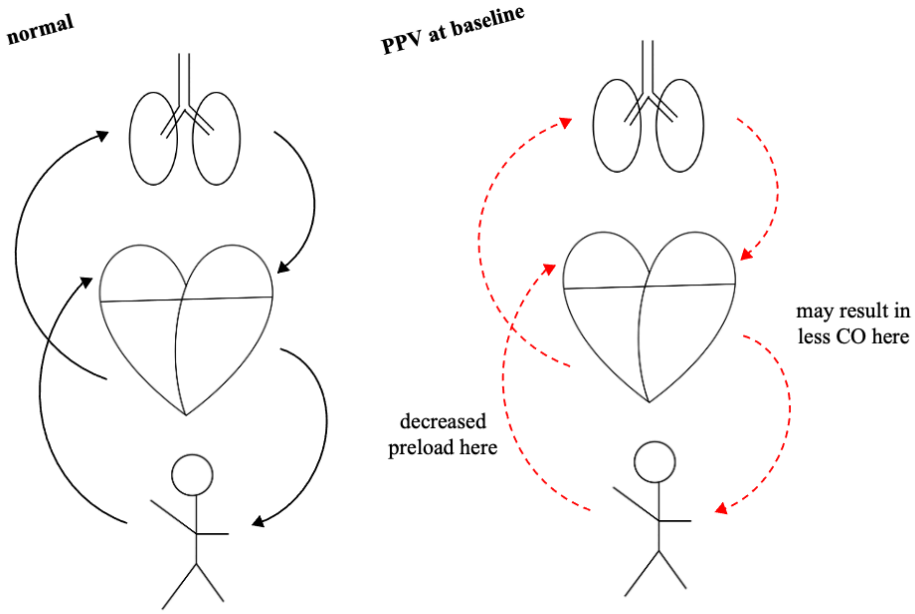
cardiac tamponade	pericardiocentesis
tension hemo/ pneumothorax	chest decompression (needle, finger, chest tube)
pulmonary obstruction (PE or pHTN)	PE: anticoagulants &/ or thrombolytics, surgical or procedural thrombolysis pHTN: pulmonary vasodilators
abdominal compartment syndrome	physical positioning, escharotomy with burns, gastric decompression, drainage of fluids, surgery, etc.
excessive AutoPEEP	longer E-time, drugs, breaking the circuit

Of note here, as it is related to vent management, would be the fact that **AutoPEEP** in and of itself can cause hypovolemia and an obstructive shock state. So by extension bronchodilators and the other **Medications for Obstruction** could fall into this discussion. Same goes for pulmonary hypertension, which is treated with pulmonary vasodilators as discussed shortly in **Others Meds in Mechanical Ventilation**.

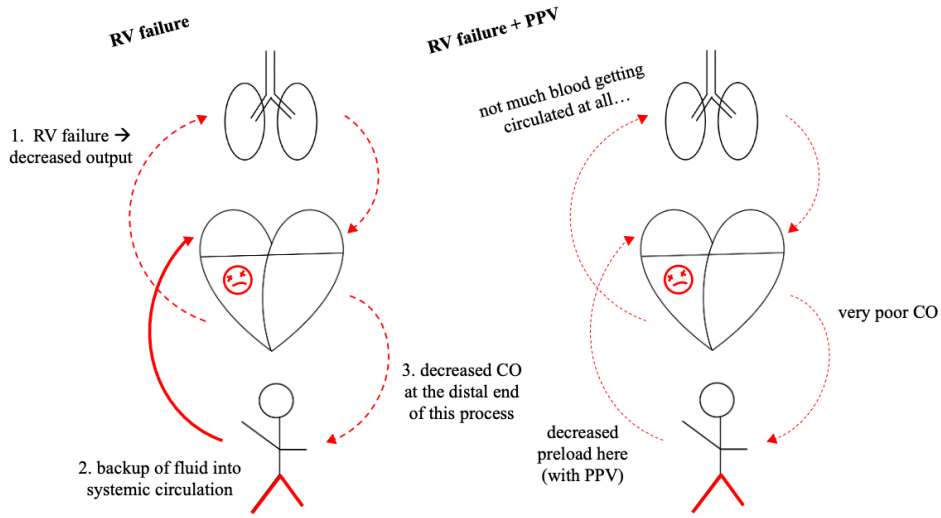
Now lastly we have cardiogenic shock. Specifically, medications to address a state of cardiogenic shock with specific consideration(s) for mechanically ventilated patients. In general, the choice of a vasoactive medication (which includes pressors, inotropes, chronotropes, etc.) doesn't vary based on whether or not the patient is intubated and/ or being ventilated or not. That said, there is a distinction between right-sided and left-sided cardiogenic shock and that does affect the degree to which mechanical ventilation can have an adverse effect on the patient.[527] In very broad strokes, positive-pressure ventilation is more detrimental in right-sided heart failure or cardiogenic shock and may actually be beneficial, in terms of hemodynamics, with left-sided issues.

[527] Alviar & friends, 2018 - This concept is explained in great detail in this paper, "Positive Pressure Ventilation in the Cardiac Intensive Care Unit"

A very simplified way of thinking of this is that positive pressure ventilation decreases preload (i.e., blood flow into the right atrium):[528]

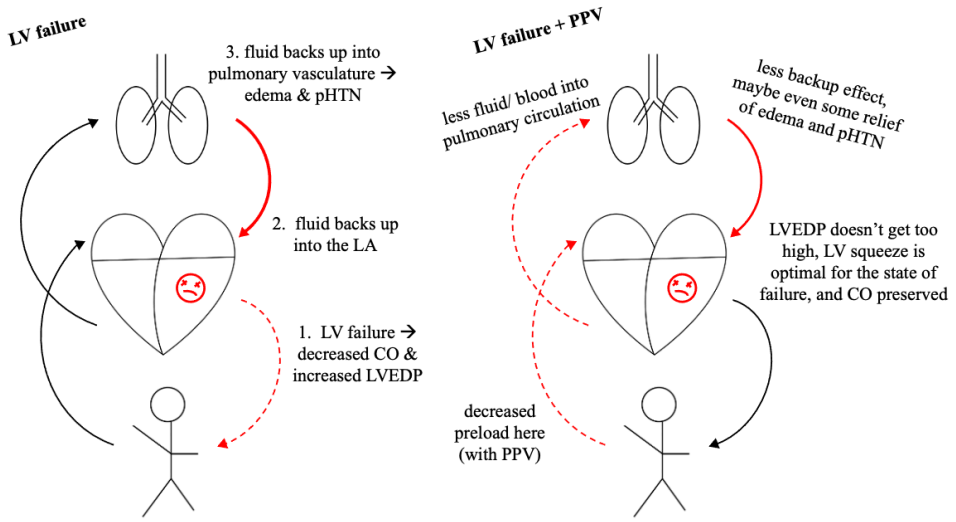


In right-sided heart failure we need preload to make it over to the left side of the heart to maintain cardiac output, so any further impediment can be catastrophic:



[528] As discussed early on in **How is Positive-Pressure Ventilation Different?**

With left-sided heart failure, however, a decrease in preload can be of benefit, as fluid is already backing up into the lungs and we want to both avoid overloading pulmonary circulation and avoid pushing too much fluid into the left ventricle (beyond its capacity to push it out with each contraction):[529]



Taking this a step further, in left-sided failure if we do allow preload to increase excessively, we get fluid in the lungs and respiratory compliance gets worse. In the spontaneously breathing patient, the result is then an increased effort to breathe, i.e., a stronger negative inspiratory force. This then increases both preload and afterload putting more stress on that failing left ventricle. Positive-pressure ventilation and mechanical ventilation decrease preload to the heart and can help prevent or reverse this vicious cycle.[530]

Drugs for Acidosis

In the section on **Acidosis** we mentioned utilizing the vent as a tool to help correct (or at least prevent the deterioration of) an acidotic state. We mentioned then that we most often do this in states of marked metabolic acidosis by simply increasing **Minute Volume**. We dived a bit deeper in **Blood Gas Analysis**, but in general practice we don't often get into the weeds and, therefore, typically stick with this strategy of simply adding more minute volume when acidosis is profound. Along with this vent strategy, however, there are other interventions that can be utilized concurrently. Some of these, such as continuous renal replacement therapy (CRRT) or hemodialysis simply aren't available in transport.[531] One that is available and is routinely discussed in all subfields of acute care medicine is the use of sodium bicarbonate.

In broad strokes, sodium bicarbonate is indicated as an infusion for marked metabolic acidosis when treatment of the underlying cause alone is insufficient or unavailable. There is some nuance to this, however, because sodium bicarb (just like any other medication) does have some adverse effects that must be taken into consideration; these include "paradoxical respiratory acidosis, intracellular acidosis, hypokalemia, hypocalcemia, alkalosis, impaired oxygen delivery, cerebrospinal fluid acidosis, and neurologic dysfunction."[532]

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[529] For a review on this graphic and how to issue similar sketches to better understand concepts of hemodynamics, refer to our blog post: Hemodynamic Sketches

[530] Yartsev, 2025c - As is almost always the case, this process is explained in much more detail and eloquently over at *Deranged Physiology*

[531] CRRT would be used for acidosis related to kidney injury; hemodialysis for acidosis related to toxins that can be removed by the process

[532] Eraky & friends, 2024 - Deep dive into the adverse effects and consequences of bicarb utilization

There are also a couple of types of metabolic acidosis in which it has been demonstrated that bicarb has no beneficial effect: diabetic ketoacidosis and mild lactic acidosis.

As for specific guidance in light of those things, we'll simply reference this summary chart:[533]

Summary: Bicarbonate in the ICU		
Give	**Don't Give**	**Consider**
Hyperkalemia**	DKA (especially pediatric)	Lactic acidosis with pH < 7.1
NAGMA	Lactic acidosis with pH > 7.15	Lactic acidosis + AKI or NAGMA
Sodium channel blocker overdose		Cardiac arrest with severe acidemia
Salicylate toxicity (alkalinization)		Uremic acidosis

**Consider clinical context — bicarbonate may be less effective if not acidemic, and its benefit may come more from the sodium load than from intracellular K^+ shifting. In some cases, hypertonic saline may be preferred. Apply bicarbonate selectively, not reflexively.

In terms of ventilation, there is the oft-cited concern that rapid administration of bicarb or administration of bicarb in the setting of inadequate ventilation can decrease the benefit or potentially cause harm. The solution is simple - don't ventilate patients with an inappropriately low minute volume. Also just give bicarb slowly.

As for other medications to treat acidosis, that list might include things to treat underlying causes - insulin and fluids in diabetic ketoacidosis or specific antidotes for certain toxins, etc. That said, there aren't any specific concerns or consideration in terms of mechanical ventilation for those sorts of things.

Pharmacology for Lung Injury

Treatment of **Lung Injury** and ARDS often includes glucocorticoids (discussed above in **Medications for Obstruction**) and may include paralytics (also discussed above, but in **Agents for Comfort and/ or Synchrony**). It is also quite common to see these patients treated concurrently for infection or sepsis with antibiotics, but the lung injury component often persists beyond the acute infection. And then beyond that there isn't a lot we routinely do pharmacologically for ventilated patients. That said, it's worth mentioning a few things that have been tried and are still used in some places.

Most prominent are the inhaled pulmonary vasodilators. This includes things like inhaled nitric oxide (iNO) or inhaled prostaglandins (such as epoprostenol, iloprost and alprostadil). The theory behind this class of medications is that they dilate pulmonary vasculature to reverse, to some degree, **Hypoxic Pulmonary Vasoconstriction**; this then results in better ventilation perfusion (V/Q) matching, relieves pulmonary hypertension and lessens right-ventricular afterload. All that said, the data to support their use in ARDS is not quite there.[534] This is not to say, however, that these effects don't occur, just that the mortality benefit in ARDS has not been demonstrated; so it very well be the case that transport crews do encounter patients receiving these medications (and because of that, we'll discuss them a bit more in the next section, **Other Meds in Mechanical Ventilation**).

Other agents that have been used and/ or studied, but don't have robust evidence or widespread adoption include surfactant, beta-agonists and Vitamins C & D.[535] There is also ongoing research for additional therapeutics that may become more prevalent in the future. And then very last thing to mention is that there are now agents that are used in cases of lung injury specific to COVID-19, but most of these are pretty niche and are not things routinely carried by transport crews.[536]

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[533] Rachel F, 2025 - Show notes for a *PulmCast* episode that reviews utilization of bicarb in the ICU setting
[534] Fielding-Singh & friends, 2018 - This paper evaluates the support (or lack of support) for a number of different strategies in the treatment of ARDS
[535] Lewis & friends, 2019 - Review of the various treatment strategies for ARDS and the current evidence for each
[536] IDSA, 2025 - More on current recommendations for COVID-19 can be found here

Other Meds in Mechanical Ventilation

Just to wrap this section up, we'll discuss a few medications and classes of medications that don't fit cleanly into the preceding sections: inhaled pulmonary vasodilators & diuretics.

Starting with the inhaled pulmonary vasodilators: these are medications that, as one might assume per the name, cause vasodilation in the pulmonary vasculature. This can be clinically helpful in two distinct situations: marked hypoxia and increased pulmonary vascular resistance (PVR). For hypoxia, these medications selectively vasodilate aerated lung areas to improve V/Q match.[537] Increased PVR, on the other hand, can cause or worsen right-sided heart failure; reducing that resistance or afterload can lessen strain on the right ventricle and improve outcomes. Other less frequent uses for inhaled pulmonary vasodilators would be in the treatment of pulmonary embolism or right-sided myocardial infarction.[538]

Inhaled nitric oxide (iNO) and inhaled epoprostenol (iEPO) are the two most common agents.[539] iNO and iEPO have comparable efficacy, but iNO has traditionally been more common in hospital settings. That said, it requires some specialized equipment (that isn't generally available for transport), is expensive, and can cause rebound pulmonary hypertension (pHTN) with discontinuation of the therapy. iEPO, on the other hand, can be given via a jet nebulizer[540] (similar to what we'd use for albuterol, just a certain type - discussed shortly), is cheaper, and may not cause as profound of a rebound pHTN.[541] For that reason, iEPO is becoming more common in the transport setting.

At the moment we aren't aware of any iNO setups that are routinely used in transport. Most delivery systems are quite large and would be difficult to move into a vehicle, but it is within the realm of possibility so we'll review the dosing details briefly. iNO is dosed in parts per million (ppm). Normal range is up to 20 or 40ppm, but systems can commonly deliver more than that.[542] Two things to note on iNO before moving on. First is that delivery of iNO involves both oxygen (O_2) and nitric oxide (NO) and this combination results in the production of some nitrogen dioxide (NO_2).[543] NO_2 can cause bad things to happen in the lungs and airways.[544] For this reason, some iNO delivery systems monitor and display NO_2 (in ppm) for trending purposes (i.e., so that the system can be troubleshooted or therapy discontinued if too much NO_2 is observed). Second thing to note is that discontinuation of iNO should be done slowly because rebound pulmonary hypertension can occur (as mentioned above).

Inhaled epoprostenol (again, iEPO) is more commonly known by one of its trade names, Flolan.[545] It can be administered in an inhaled/ nebulized form similar to how beta-agonists are administered using small-volume nebulizers (SVN). The difference here is that we use a large-volume nebulizer (which could be referred to as an LVN, but is commonly known by the specific brand MiniHEART) which is similar to the SVN, but has some features that make it better than the standard nebulizer. First is that it often has a valve that limits

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[537] An important note here is that this refers to *inhaled* pulmonary vasodilators, systemic agents can actually worsen V/Q matching because under-aerated areas will also vasodilate; refer back to **Hypoxic Pulmonary Vasoconstriction** for a review of this general concept

[538] Farkas & Thomas, 2020 - All of these concepts are discussed in this podcast on pulmonary vasodilators

[539] Rajagopal & friends, 2023 - This overview is focused on the treatment of pHTN in non-cardiac surgery specifically, but it does provide a broad overview of the concepts, pathophysiology and agents used

[540] Dolovich & Dhand, 2011 - The term "jet nebulizer" simply means that it used compressed gas to disperse the liquid, it can also be called a "pneumatic nebulizer;" more on all the various types of devices and the associated terminology in this paper; and to review **In-line Nebulization** on the vent, refer back to that section

[541] Regarding the difference in rebound pulmonary hypertension with discontinuation of iNO vs. iEPO: it can occur with both, but there is a larger body of evidence on this effect with iNO; also the mechanism by which it occurs in either case is different and seems to point to a larger/ greater affect (i.e., worse/ greater degree of rebound) with iNO; and lastly there is a longer half-life with iEPO versus iNO which could contribute to this effect [and then rather than citing a million papers here, we'll simply attach this discussion and response with OpenEvidence for those interested in further reading on all of this]

[542] Rajagopal & friends, 2023 - Cited above, see here for a brief overview of dosing for both iNO and iEPO

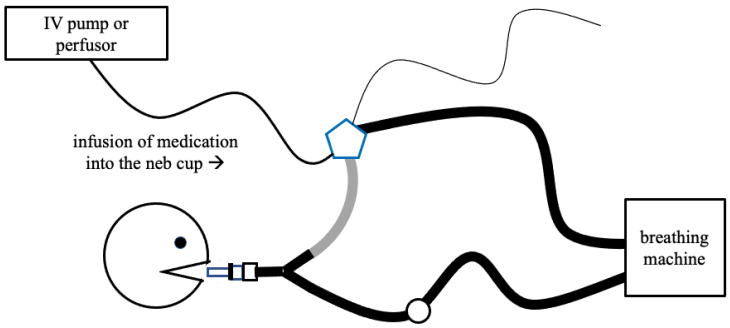
[543] And while we're on this point, also note that nitrous oxide or "laughing gas" is another thing altogether (N_2O)

[544] Petit & friends, 2017 - More on the effects of nitrogen dioxide and why it occurs in iNO therapy here

[545] Farkas, 2024b - It actually has a few more names, as noted in this general overview of pulmonary vasodilators from the *Internet Book of Critical Care* - prostacyclin or prostaglandin I2

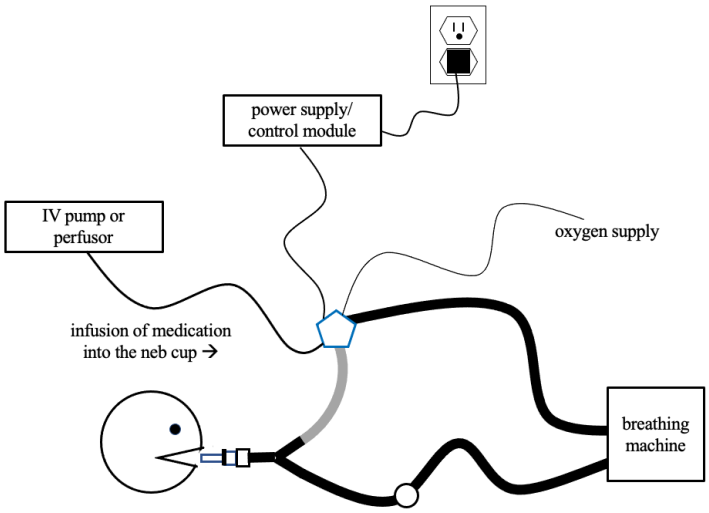
waste of the medication on exhale (i.e., medication is only delivered on inhale).[546] Second is that it can hold a larger volume of medication (commonly 30ml capacity). Third is that it can deliver smaller particle sizes of nebulized medication, which theoretically improves medication delivery. And fourth is that it is calibrated to give a certain amount of medication (ml/hr) with a given flow of oxygen into the device (2lpm).[547]

The setup would be the nebulizer device with an oxygen supply as normal and then an infusion running into the thing - just note that this requires a point on the cup to which a medication infusion can attach and not all nebulizers have this functionality:[548]



Now we could, in theory, use a simple SVN, but those devices are not typically manufactured to give predictable output, as the idea is that dose is limited by the volume of drug put into the nebulizer cup. We could also use a nebulizer device without a medication port for the continuous infusion, it would just require a bit more monitoring to ensure the cup doesn't run dry (and obviously re-filling it before it does get empty).

And then as for the Aerogen device, which uses vibrating mesh technology, the setup has the same general components, but the way in which the droplets/ particles are created is unique. It basically involves liquid medication instilling across a disc with lots of little tiny holes in it; as that disc vibrates (hence the power supply here), particles are released into the circuit:[549]



[546] This is not a feature unique to the MiniHEART, rather it is more common with these types of devices than with normal nebulizers
[547] While we could, in theory, do an experiment to see what flow (lpm) correlates with what output (ml/hr) with any nebulizer that isn't done for SVNs because the idea is that we limit dose by the total amount of medication into the reservoir/ cup
[548] Nelson, 2021 - YouTube video that shows this same setup
[549] Aerogen, 2011 - very brief YouTube video from Aerogen to show how this works

And there is data to suggest that this delivery method (vibrating mesh technology) yields better medication delivery over standard jet or pneumatic devices.[550]

Regardless of which delivery mechanism is used, dosing is important. iEPO can be given up to 50ng/kg IBW/min; some places will start at 50 and titrate down, others start low and adjust up as needed. And then to actually give a desired dose, this depends on which setup will be utilized. If using a MiniHEART (or other comparable pneumatic device) we have to prepare the medication so that delivery at 8ml/hr (with 2lpm of flow into the device, as that's what it has been manufactured to do) will give the desired dose.[551] With the Aerogen, on the other hand, delivery is about 0.38ml/min or 23ml/hr.[552]

Important takeaway here: if a patient is being treated with iEPO using the Aerogen or a similar device, we can't simply swap it over to a MiniHEART or other LVN, jet nebulizer because the delivered dose will almost always be different - each setup requires a specific concentration or preparation of iEPO. For that reason, it is sometimes necessary to transport the device with the patient.

And then following along with this idea: if we do go to transport one of these patients undergoing treatment with an inhaled pulmonary vasodilator (iNO or iEPO) and we cannot continue therapy for whatever reason, the risk exists that abrupt discontinuation will lead to rebound pulmonary hypertension and, therefore, subsequent hypoxia and maybe right-sided heart failure. It is probably OK to transition from iNO to iEPO, if possible, but it is unlikely that iEPO lives in a given transport service's med bag/ formulary. Another option would be to consider inhaled nitroglycerin.

Inhaled nitroglycerin (nitro) functions as a pulmonary vasodilator because it gets converted to NO.[553] Dosing has traditionally been done in mcg/kg/min which would require some sort of delivery system like the MiniHEART or Aerogen, but studies have shown that a bolus dose can be used as a stopgap measure. The half-life of nitro is longer than that of both iNO and iEPO and it seems that the duration of action following a bolus dose is somewhere in the range of 20-30 minutes. Theoretically, nebulizing 5mg of nitro in an SVN could maintain the pulmonary vasodilation/ prevent rebound pulmonary hypertension (that occurs via vasoconstriction) for a bit of time. For prolonged transport, this could simply be repeated either as needed or at fixed intervals every 20-30 minutes.

And then to make this happen, it depends a bit on what concentration/ preparation of nitro is available on hand. Some services carry more-concentrated preparations, such as 50mg/10ml (5mg/ml), which would simply require adding 1ml to a few ml of saline or sterile water in the SVN. Others carry a more dilute, less-concentrated preparation, such as 25mg/250ml (100mcg/ml), which would necessitate 50ml of liquid - this could be better delivered simply by a continuous neb treatment (i.e., refilling the cup as it gets low throughout transport). In any case, just know that this is simply a bridge to resumption of either iNO or iEPO after transport.

If we have a patient on iNO or iEPO and can't continue the therapy and also cannot nebulize nitroglycerin on the way, one other option could be inhaled milrinone.[554] Milrinone used in this way has similar efficacy to nitro,[555] but may have a longer duration of action.[556] It could be as simple as asking the sending

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[550] Salemeh & friends, 2025 - This systematic review looked at vibrating mesh technology vs. jet nebulizers in the management of asthma & COPD

[551] And it very well may be that there are other devices on the market (now or in the future) that give different admin rates (ml/hr) with the same flow (lpm) or that require different flow (lpm) for the same admin rate (ml/hr), it's just that this seems to be the most common

[552] Aerogen, 2024 - This is the product manual/ instructions for the Aerogen that cites this specific flow rate

[553] Farkas, 2024b - More on this idea of inhaled nitroglycerin for use a pulmonary vasodilator in this chapter of the *Internet Book of Critical Care* that we cited above; the section on nitro cites five studies that go into further detail, pdfs for all are linked from the reference page

[554] Yartsev, 2023e; Farkas, 2024b - Read all about milrinone over at *Deranged Physiology*; it is also discussed briefly in the *Internet Book of Critical Care* on their page discussing inhaled pulmonary vasodilators

[555] Singh & friends, 2010 - This paper compared inhaled preparations of nitro and milrinone to one another for pediatric patients with pHTN due to congenital heart disease

[556] Couture & friends, 2019; Wang & friends, 2009; Kundra & friends, 2018 - This first study proposed dosing milrinone every four hours when used in combination with iEPO; the second study, however, suggests effects only persist thirty minutes after cessation of a

facility to administer a large nebulized bolus dose (given with a normal SVN) while the patient is prepared for transport[557] - that could theoretically buy time to get the patient where they need to go and resume whatever other pulmonary vasodilator therapy they had going beforehand. That said, this is rarely done, the evidence isn't awesome, it most likely warrants a conversation with one (or both) of the intensivists on either end of the transport, and the dose would probably need to be in excess of 5mg or 50mcg/kg.

And then very last thing to mention on inhaled pulmonary vasodilators is that there is an agent called levosimendan that could also be used and that has a demonstrably long duration of action - up to three hours.[558] That said, it is unavailable in the United States and therefore would only be an option overseas.

Quick summary chart of the inhaled pulmonary vasodilators that we've mentioned before we move on:

agent	pros	cons
iNO	most widely used; dosing is simple	refractory pHTN with cessation; requires specialized and bulky equipment
iEPO	easy to use in transport (so long as a device is available)	refractory pHTN with cessation; must be prepared specifically based on delivery method; requires specialized equipment
nitro	readily available to most EMS crews; can be given with SVN	short duration of action; concentration varies
milrinone	could theoretically be given as a bolus dose at start of transfer; can be given with SVN	short(ish) and questionable duration of action
levosimendan	long duration of action, could theoretically be given as a bolus dose at start of transfer; can be given with SVN	not available in the US; also not a ton of data at this time

This is not, of course, a comprehensive list, rather just an overview of the ones most likely seen in the transport setting. There are more inhaled agents we didn't get into, as well as oral agents that can be used in combination with inhaled agents, and then sometimes we see intravenous infusions of things (including EPO and milrinone) instead of inhaled delivery.[559]

Next on our list are the diuretics. The gist here is that too much fluid in the system can result in worse outcomes and prolonged time on the ventilator. That said, it is often the case that patients need fluids during the resuscitation phase of care. Once the patient has stabilized, however, the process of deresuscitation and removal of excess fluid begins. While it isn't super common that patients at this phase of care are in need of critical care transport, there are some cases in which it does occur.[560] Also diuretics, as a class of medications, are quite common in critical care, so it makes sense to review them briefly while we're on the subject of medications.

In terms of interaction with and complications related to mechanical ventilation, the main thing is that diuresis can lead to hypotension. Or rather, excessive diuresis or excessively fast diuresis can lead to hypotension. This combined with the fact that mechanical ventilation in and of itself can do the same means that the patient may be at increased risk for hypotension. Now if a patient is at the deresuscitation phase of their care, it is unlikely that vent changes will be made to further decrease blood return to the heart (i.e., we likely won't be titrating up on **PEEP** or increasing **Respiratory Rate** to any significant degree). That said, it could be that a patient is being weaned from the vent (i.e., doing a spontaneous breathing trial) while undergoing

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continuous neb (but perhaps a large, bolus dose would last longer); the third also cites duration of action around thirty minutes with a bolus dose of 50mcg/kg (so that'd be just shy of a 5mg bolus dose for the average-sized human)

[557] And milrinone often comes in 1mg/1ml preparations, so this would simply mean 5ml of the medication in the SVN

[558] Kundra & friends, 2018 - Cited above, this study compared inhaled milrinone to levosimendan

[559] Yartsev, 2022a - General overview of pulmonary vasodilators (not just the inhaled ones); an example of an oral agent would be sildenafil (Viagra), which is actually the same general/ broad class of medication as milrinone (phosphodiesterase inhibitor; but they fall into different sub-types, so the similarity isn't super close)

[560] The best example would be after damage control or emergent surgery

diuresis; if we then show up and place the patient back on a rate for transport (i.e., put them in **Assist Control** or **SIMV**), we could see a precipitous drop in blood pressure.

Now rather than get into all the specifics of various diuretics, we'll simply provide this summary chart for name recognition and key facts:[561]

drug class	examples	administration	notes
loop diuretics	furosemide (Lasix) bumetanide (Bumex)	IV bolus or infusion, also can be PO	most often first-line, can cause electrolyte imbalances
potassium-sparing diuretics	amiloride	PO with relatively rapid onset	can cause electrolyte imbalances
thiazides	chlorothiazide metolazone	IV (chlorothiazide) PO (metolazone)	can cause electrolyte imbalances
SGLT2 inhibitors	dapagliflozin empagliflozin	PO	long list of contraindications…
carbonic anhydrase inhibitor	acetazolamide	slow IV bolus	used with metabolic alkalosis, can cause electrolyte imbalances
mineralocorticoid antagonist (also potassium-sparing)	spironolactone	PO with slow onset (a few days to reach therapy)	slow onset (see left), can cause electrolyte imbalances
hypertonic solution	3% saline	slow IV bolus	rescue option used in combination with others

One final thing to mention here is that discontinuation of diuretics doesn't generally cause an abrupt rebound in fluid overload or anything like that, so if we're out of IV pumps/ perfusors or a specific medication is beyond our scope of practice, it is probably OK to simply discontinue the therapy en route and have it restarted afterwards.

[561] Farkas, 2025a; Yartsev, 2024b - Both pages discuss the concept of diuresis and the specific diuretics used in critical care

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Suggestions for Further Study

Just some guidance based on what kind of medium someone is looking for. This is not an exhaustive list, but just some places to start for getting better at the management of ventilated patients. Also recognize that each of these references has way more to offer than just the specific content linked, browse them all for more intel on many of the things we've discussed in this manual.

text, eBook

Basic Principles of Mechanical Ventilation by Melody Bishop, BSc, RRT, CCAA

Principles and Practice of Mechanical Ventilation by Martin J. Tobin (3rd edition)

text, web-based

Deranged Physiology, section on Respiratory

RebelEM, Simplifying Mechanical Ventilation, 6-Part Series

video, vent specific

Strong Medicine Series on Mechanical Ventilation

Hospitalista Series on Mechanical Ventilation

video, physiology

Ninja Nerd Science, section on Respiratory

Khan Academy, section on Advanced Respiratory System Physiology

audio/ podcast

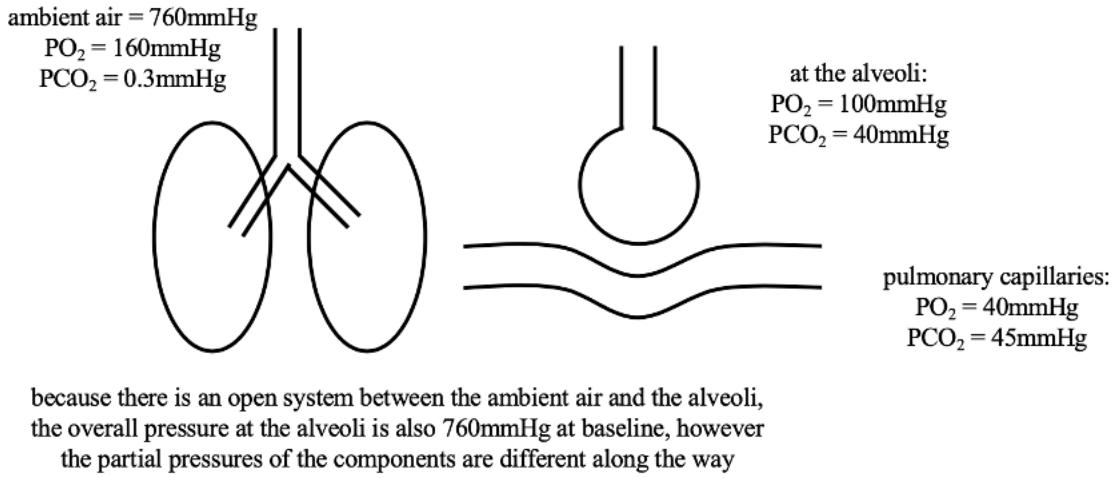
EMCrit Dominating the Vent, 2-Part Series

FlightBridgeED Vent Series, 3-Part Series

Appendix

Alveolar Gas Equation

The alveolar gas equation allows us to calculate the partial pressure of oxygen in the alveoli in a given set of circumstances. We used this equation to get values listed in some of the graphics throughout this manual:



The equation goes like this:[562]

$$PAO_2 = FiO_2(P_{atm} - P_{water}) - (PaCO_2/RespQ)$$

PAO_2 is partial pressure of alveolar oxygen
FiO_2 is fraction of inspired oxygen, 0.21 for ambient air
P_{atm} is atmospheric pressure
P_{water} is partial pressure of water vapor at the alveoli, 47mmHg at sea level
$PaCO_2$ is as measured by blood gas (or approximated from $EtCO_2$), we'll say 40mmHg
RespQ is respiratory quotient and is assumed to be 0.8[563]

given that RespQ = 0.8, we sometimes see the equation simplified as so:

$$PAO_2 = FiO_2(P_{atm} - P_{water}) - 1.25(PaCO_2)$$

and since P_{atm}, P_{water}, and $PaCO_2$ are all held constant in our thought experiments:

$$PAO_2 = FiO_2(760 - 47) - 50$$
$$PAO_2 = FiO_2(713) - 50$$

but back to our original equation:

$$PAO_2 = FiO_2(P_{atm} - P_{water}) - (PaCO_2/RespQ)$$
$$PAO_2 = 0.21(760 - 47) - (40/0.8)$$
$$PAO_2 = 100mmHg$$

[562] Yartsev, 2024a - He's got a good graphic that shows the alveolar gas equation with all parts labeled, maybe makes a bit more sense to the visual learners than how it is represented here
[563] Patel & Bhardwaj, 2018 - Describes the detail behind this respiratory quotient idea; maybe not relevant to our discussion of vent stuff, but good nerdy details for those who want more (another option would be to find an exercise physiology textbook, likely to be some good stuff there)

Other iterations of the alveolar gas equation that we demonstrated in the manual are shown here:

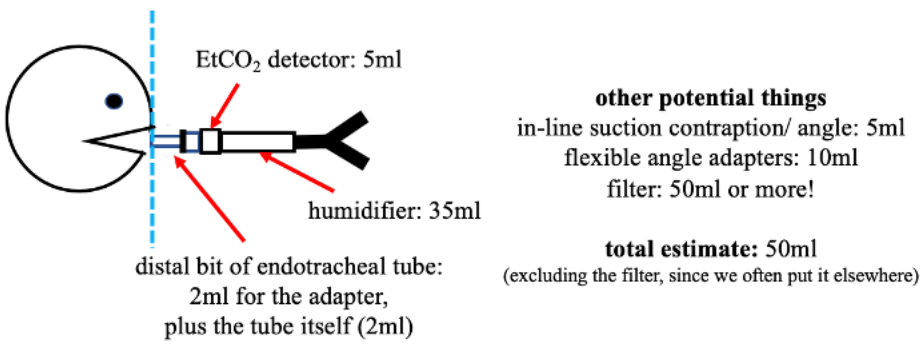
PAO_2 at 100% or FiO_2 1.0 (no PEEP)
$PAO_2 = FiO_2(760 – 47) – 50$
$PAO_2 = 663mmHg$

PAO_2 with 5cm PEEP (room air)[564]
$PAO_2 = FiO_2(760 (+ 4) – 47) – 50$
$PAO_2 = 101mmHg$

So we can use the alveolar gas equation to solve algebra problems in an effort to show how things like **Fraction of Inspired Oxygen** and **Positive End-Expiratory Pressure** affect PAO_2. And then if we know how much oxygen should be getting to the alveoli and can measure how much oxygen made it into the arteries (PaO_2 with **Blood Gas Analysis**), then maybe we can understand something about the efficacy of that exchange. To say it another way, the idea is that we can use values for PAO_2 and PaO_2 to inform us on what is going on with a patient in reference to the movement of oxygen from the input of our vent system into the bloodstream. Values like A-a Gradient and a/A Ratio attempt to do just that. Now there are some limitations to both of these values and their application may be limited in the transport setting, so we won't get into the details here.[565]

Mechanical Dead Space

In order to determine the effect of mechanical dead space, we first need to know how much volume each of the extra components takes up. This varies a lot depending on which specific devices we use and can be found on product labels, but we'll just generalize it here:



[564] Just a friendly reminder that $5cmH_2O$ is roughly 4mmHg, see chart in **Measuring Pressures**
[565] Strong, 2014; Yartsev, 2024c - The first is a video that explains A-a gradient; the second is an article that discusses these types of measurements and identifies issues with their application to clinical practice

Let's say we want to figure out to what effect 50ml of added **Dead Space** impacts **Ventilation** in our patients. Now this gets a little weird and the math takes a few leaps of faith along the way, but let's follow along and then compare what we come up with to data after the fact. Also, note that we are going to introduce a few new ideas here and that we will get more into those in the very next section:[566]

assume a patient of 65kg IBW
being ventilated at TV 6ml/kg (390ml) and RR of 17
MV calculated = 6630ml/min

now we already said a few things about this:
alveolar TV = TV – anatomic dead space
and this anatomic dead space is approximately ⅓ of TV
so alveolar TV = 390ml – (390ml x ⅓)
alveolar TV = 390ml – 130ml
alveolar TV = 260ml
VA = RR x alveolar TV
VA = 17/min x 260ml
in this case VA = 4420ml/min

and if we add 50ml more of dead space into the situation
alveolar TV = TV – anatomic dead space – mechanical dead space
so alveolar TV = 390ml – 130ml – 50ml
alveolar TV = 210ml
VA = RR x alveolar TV
VA = 17/min x 210ml
in this case VA = 3570ml/min

We already know that there can be a discrepancy between these two versions of alveolar minute volume, the one with mechanical dead space left out and the one with it included. But now let's consider the idea that the amount of carbon dioxide produced per minute doesn't change from case to case, rather it's simply the case that less of that of it gets exhaled. So how much carbon dioxide gets left behind in the system and what kind of effect does that have on the body? To answer the first question, let's look at the following relationship:

$$\frac{V_D}{TV} = \frac{EtCO_2 - PECO_2}{EtCO_2}$$

[566] And to review these ideas: **Ideal Body Weight**, **Tidal Volume**, **Respiratory Rate**, and **Minute Volume**

There are two versions of this formula that use $PACO_2$ and $PaCO_2$ rather than $EtCO_2$, but it has been proposed that this representation might be of value in calculating dead space in practice.[567] So simply for the sake of this example, we will go with that. Now that $PECO_2$ value is something we haven't discussed yet; it is the mean partial pressure of carbon dioxide during exhalation. A normal value is around 30mmHg and it could also be calculated based on the idea that a normal fraction of expired carbon dioxide ($FeCO_2$) is about 4%:[568]

$$PECO_2 = FeCO_2\ (P_{atm} - P_{water})$$
$$PECO_2 = 4\%\ (760\text{mmHg} - 47\text{mmHg})$$
$$PECO_2 \approx 28.5$$

now if we use that value and the previous equation,
we can solve for an expected $EtCO_2$ in either of the dead space cases in question

only anatomic dead space:

$$\frac{130}{390} = \frac{EtCO_2 - 28.5}{EtCO_2}$$
$$EtCO_2 \approx 43$$

with mechanical dead space added in:

$$\frac{180}{390} = \frac{EtCO_2 - 28.5}{EtCO_2}$$
$$EtCO_2 \approx 53$$

A difference in $EtCO_2$ of 10mmHg doesn't necessarily mean that a corresponding quantity of carbon dioxide remains in the blood and impacts the body. The purpose of this exercise was simply to show that the potential exists for a buildup of carbon dioxide in the alveoli. In the transport setting where $EtCO_2$ monitoring is routinely used to assess ventilation, we would simply increase **Minute Volume** to bring that second value into a normal range. But let's suspend that idea for just a moment longer and consider what impact this might have if we failed to do that. Researchers looked at this very problem and determined that removing 115ml of dead space from a circuit resulted in a decrease in $PaCO_2$ of 11mmHg and an increase of pH from 7.30 to 7.38.[569] Furthermore, they were able to do that with less minute volume. Now this was in patients with ARDS in which one of our concerns is the amount of air needed to maintain ventilation and consequences of that air on the patient's pulmonary system, but the findings are pretty significant.

Back to our discussion and application to the transport setting: we said just a moment ago that we could potentially avoid this increased carbon dioxide retention by monitoring $EtCO_2$ and increasing minute volume to accommodate, but the truth is that doing so isn't always a benign thing. Going up on **Tidal Volume** or **Pressure Control** will increase pressure (**Plateau Pressure** and **Driving Pressure**), while going up on **Respiratory Rate** has the potential to cause discomfort and increase that percentage of time at decreased preload (%TaDP) concept.[570] If we can promote carbon dioxide removal while simultaneously avoiding all of those things, this seems like a pretty good reason to be conscious of adding unnecessary things into the vent circuit whenever possible.

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[567] Siobal, 2016 - This is a theoretical thing and would require further experimentation, but it serves the purpose of showing to what extent dead space might impact quantitative measures of $EtCO_2$, with all other things being equal
[568] ScyMed, 2018 - Good reference for calculations and normal values for all things physiology
[569] Hinkson & friends, 2006 - Even though the study had a small sample size, the findings support that support this idea of limiting mechanical dead space
[570] Refer back to **Comfort** and **Hypotension** to review these ideas

One last thing about all of this with regards to pediatrics and volume control ventilation. We mentioned way back when that it's OK if our calculated minute volume is larger than our goal minute volume because of some complications posed by dead space.[571] We want to revisit that to show why that is and how we can mitigate it all. The example was a 4-year-old kid of 18kg:

TV = 6 – 8ml/kg IBW
TV = 6 – 8ml/kg x 18kg
TV = 108 – 144ml

MV goal = 100ml/kg IBW/min
MV goal = 1800ml/min
MV goal = 1.8L/min

MV calculated = RR x TV
MV calculated = (20 – 28)/min x (108 – 144)ml
MV calculated = 2160 – 4032ml/min
MV calculated = 2.2 – 4L/min

Just as with the adult patient, we have anatomic dead space that is always there and then mechanical dead space that we add in. But we never did consider that the vent tubing itself has some flex to it. If we look closely at the label of our vent tubing, it may say something like "compliance 0.0008L/cmH_2O." So let's take that hypothetical example and run with it:

we'll go with a TV of 6ml/kg (108ml) and a RR of 24
MV calculated = 2592ml/min
VA = RR x (TV – dead space)

to summarize all the dead space components:
we know we have about 36ml (1/3 of TV) anatomic dead space
let's say 20ml of mechanical because we have a pedi humidifier and $EtCO_2$ detector
and let's assume a driving pressure of 12cmH_2O to get to our TV goal
0.0008L/cmH_2O x 12cmH_2O = 10ml
total dead space = 36ml + 20ml + 10ml
total dead space = 66ml

VA = 24/min x (108ml – 66ml)
VA = 1008ml/min

Now in this case the alveolar minute volume is low (minute volume goal was 1.8L/min), but we could then look at exhaled tidal volume and $EtCO_2$ to titrate up to an appropriate level. But what if this had been a 10kg two-year-old?

TV 6ml/kg = 60ml
total dead space = 66ml
which mathematically means no actual ventilation!

[571] We first mentioned this consideration (of dead space with pediatric patients) in **A General Vent Strategy**

Just to be clear, this isn't completely the case. As tidal volume decreases we likely get less anatomic dead space as airway structures don't flex and expand as they normally would with the delivery of normal-sized breaths. We mentioned before that we assume dead space is constant when going up on tidal volume, but there is some variation here and it is most notable at the extreme end of low.[572] Knowing to what degree this type of thing happens isn't generally possible in transport, but the takeaway point still stands – be sure to consider these things when ventilating in volume control with small volumes.

One last thing to consider is the idea that if we are using uncuffed endotracheal tubes with our kids, some tidal volume may get lost as air moves back past the tube to the oropharynx.[573] So the moral of the story here is that we should either ventilate these patients in pressure control (to bypass this vent circuit stretch dead space concept) or start at a higher end of normal tidal volume and be ready to quickly go up on minute volume as soon as initiating ventilation in volume control (based on exhaled tidal volume and $EtCO_2$). As we said before, there is no right or wrong to this, so long as we know the consequences and correct actions associated with whatever choice we make.

[572] Yartsev, 2023a - We mentioned this idea that dead space does vary with tidal volume delivered back in both **Dead Space** and **Ventilation**; this same article also explains that this relationship is not constant across a body's capacity and that there seems to be less dead space with smaller tidal volumes

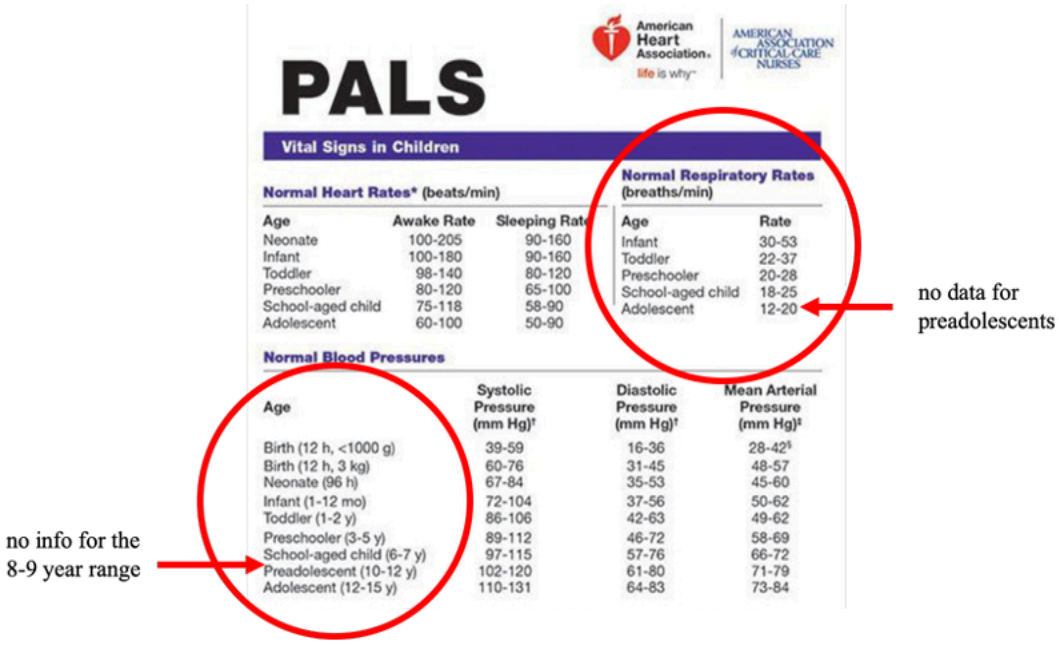
[573] Chambers & friends, 2018 - For more information on cuffed versus uncuffed tubes with pediatrics, take a look at this paper

Age-Based Settings

In an effort to make recommendations about vent settings for specific age groups, specifically **Respiratory Rate** and **Inspiratory Time**, here's how the process went:

1. Make assumptions:
    a. Normal Respiratory Rates as outlined by PALS are good enough to work with
    b. Normal respiratory rate range for an adult is 12 - 20 (cited in many, many sources)
    c. A normal I:E ratio at rest and spontaneous respiration is 1:2, but we often work with a ratio of 1:3 for ventilated patients[574]

2. Fill the gaps in the PALS Normal Respiratory Rates data set:
    a. What gaps?[575]



PALS

American Heart Association. life is why

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)‡
Birth (12 h, <1000 g)	39-59	16-36	28-42§
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	89-112	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

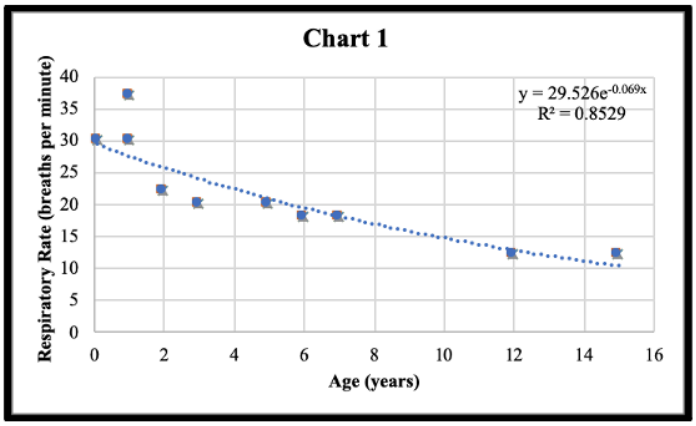
no data for preadolescents

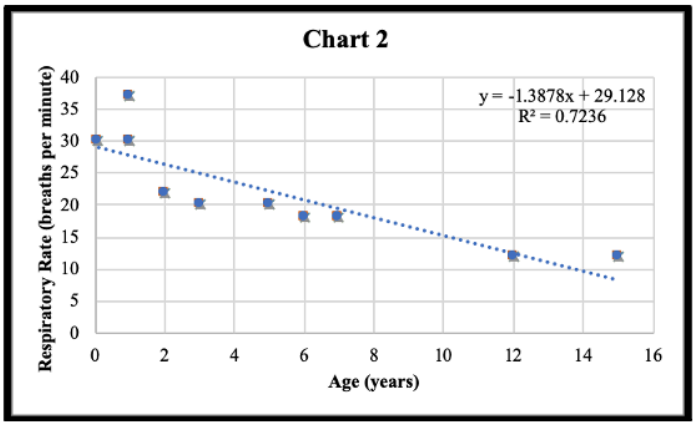
no info for the 8-9 year range

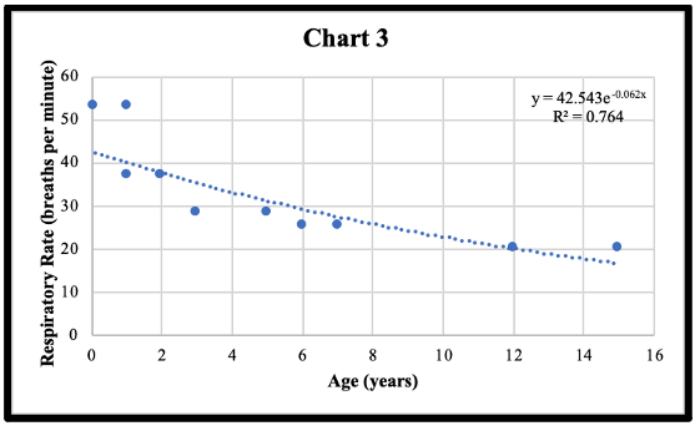
[574] And this may be by convention of leaving inspiratory time set at a given value, not necessarily because that's the thing we ought to be doing; but regardless, we'll get a range of possible values using both 1:2 and 1:3

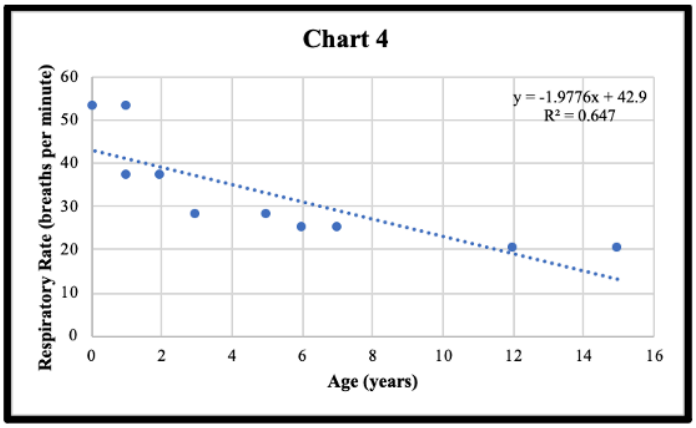
[575] American Heart Association, 2016 (image) - And we said already (section on **Respiratory Rate**) that we chose to use these values not because they are intended for use with vent management, but because they represent normal values by age and are from a reference that most of us are familiar with and have access to

b. Plot the existing data using both high and low ends of rate by age, make charts, then add lines of best fit:









c. Using the better fits (exponential regression), solve for the missing data points in the PALS chart, then add those values into a new chart (noted in blue): [576]

age description	age (years)	rate
infant	0.083 (1 month) - 1	30 - 53
toddler	1 - 2	22 - 37
preschooler	3 - 5	20 - 28
school-aged child	6 - 7	18 - 25
big kid	8 - 9	17 - 25
preadolescent	10 - 12	14 - 23
adolescent	12 - 15	12 - 20
adult	16 and up	12 - 20

[576] Range for *big kids* was calculated to be 17-26, but we went with 25 since range for school-aged child was to a max of 25 – this was an arbitrary decision, but makes the final product flow a bit better

3. Do a lot of calculations (for inspiratory times):

60s ÷ RR = time per each respiratory cycle
ex. for adult (low end RR): 60 ÷ 12 = 5s
ex. for adult (high end RR): 60 ÷ 20 = 3s

I-time = time per each respiratory cycle ÷ number of parts in that cycle
ex. for adult (low end RR, 1:2): 5s ÷ 3 ≈ 1.7
ex. for adult (high end RR, 1:3): 5s ÷ 4 ≈ 0.8

therefore I-time range for adults is 0.8 – 1.7s

4. Put all the data into a chart:

age description	**age (years)**	**rate**	**I-time (s)**
infant	0.083 (1 month) - 1	30 - 53	0.3 – 0.7
toddler	1 - 2	22 - 37	0.4 – 0.9
preschooler	3 - 5	20 - 28	0.5 – 0.9
school-aged child	6 - 7	18 - 25	0.6 – 1.1
big kid	8 - 9	17 - 25	0.6 – 1.2
preadolescent	10 - 12	14 - 23	0.7 – 1.4
adolescent	12 - 15	12 - 20	0.8 – 1.7
adult	16 and up	12 - 20	0.8 – 1.7

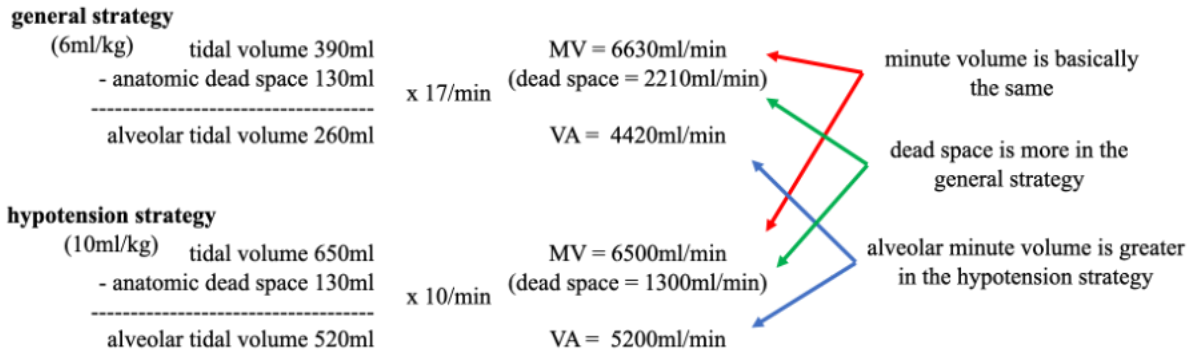
Hypotension Strategy Math

In the section where we outlined the **Hypotension** strategy, we introduced a concept which we called percentage of time at decreased preload (%TaDP). The idea was that if we decrease the overall amount of time spent pushing air into the system above our set **Positive End-Expiratory Pressure** (i.e., inspiration) then we can mitigate the exacerbation of a hypotensive state. The result was a strategy that included a shorter **Inspiratory Time**, higher **Tidal Volume**, and lower **Respiratory Rate**. We also mentioned that there are other rationales for this strategy: less **Dead Space** and lower **Mean Airway Pressure**. We are going to calculate these differences here just to give some more legitimacy to the argument.

But before we get there, one other thing to mention. PEEP is also a contributing factor to hypotension in the susceptible patient, so we want to keep that to a minimum. While it may seem like a good idea to drop PEEP to zero in the hypotensive patient (especially in light of the mean airway pressure calculations we'll show in just a moment), recognize that **Oxygenation** is also super important and PEEP is one of our tools to maintain that. Other specific benefits of PEEP that we'd like to maintain in these patients include ease of triggering spontaneous breaths and alveolar recruitment. Last thing: the positive-pressure ventilation/ PEEP → decreased preload → decreased cardiac output sequence of events can be mitigated by fluid resuscitation.[577]

Moving forward, recognize that it is totally OK to drop PEEP all the way to zero if need be, but there may be consequences and there may be other relatively simple strategies (i.e., fluids and other vent changes) to mitigate the negative consequences while maintaining the benefits. It's also just fine to drop PEEP to zero in an emergency, then work back up to a beneficial level after the acute threat has passed and other interventions have been put into place. Vent management is dynamic and we can adjust strategy as we move forward with patient care. So while we are going to show how eliminating PEEP can significantly reduce mean airway pressure, which theoretically lessens the negative consequences of positive-pressure ventilation, just know that there are multiple variables involved in this practice.

Now for the math, starting with how the lower rate, higher tidal volume strategy decreases dead space. Let's assume another 65kg patient and see how it looks. We've shown the calculations here working from the assumption that anatomic dead space doesn't change with tidal volume, but recognize that this idea is based on a number of factors and may not be the case for all situations:[578]



This demonstrates the concept that in the hypotension strategy we push less wasted air into the system. We already know that positive pressure, whether in the form of a breath being delivered or PEEP, has potential negative consequences, so if we eliminate any part of that (i.e., reduce dead space) while maintaining ventilation then our patient is better off. To say it another way, we want to try to make use (in the form of alveolar minute volume) of as much of the total air (minute volume) that we put into the system in an effort to eliminate pushing air in unnecessarily (dead space).

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[577] We mentioned this sequence of events and how to mitigate it with fluids way back in **How is Positive-Pressure Ventilation Different?**

[578] Yartsev, 2023a - To say it another way, this advantage that we calculate out is the best case scenario; we talked about this in **Ventilation** and referenced this same article then; also refer back to **A General Vent Strategy** to review that idea

The next concept to discuss is mean airway pressure. The airways and lungs live inside the thoracic cavity, so if we put pressure into the respiratory system then we see changes to pressure in the thoracic cavity. The idea is that mean airway pressure directly correlates with a concept called intrathoracic pressure and intrathoracic pressure, in turn, is the thing that causes all those hemodynamic changes associated with positive-pressure ventilation.[579] Now it gets exponentially more complex than that, as pressure at specific components within that thoracic cavity, all of which are tied to hemodynamic function, vary significantly (in terms of influence on function, not necessarily quantitatively), but the simple interpretation of the idea is that pressure we put in via the vent can disrupt hemodynamic function and result in less cardiac output. So theoretically, if we limit mean airway pressure we can minimize these potential negative consequences.

Mean airway pressure is normally measured by the vent itself, but there is a formula to estimate it using values for inspiratory time, **Peak Inspiratory Pressure**, and PEEP (and also T_{total}, which is the amount of time per breath or 60s ÷ respiratory rate):

$$P_{aw} = 0.5 \times (PIP - PEEP) \times (I\text{-time}/T_{total}) + PEEP$$

Using this formula, we built a spreadsheet of possible mean airway pressure (P_{aw}) data points for each strategy with different values for peak inspiratory pressure (PIP) and PEEP. And just to clarify, this is with an inspiratory time of 1.0s in both cases, a rate of 17 per the general strategy, and a rate of 10 per the hypotension strategy:[580]

general strategy							
P_{aw}		PIP					
		10	**15**	**20**	**25**	**30**	**35**
PEEP	**0**	1.42	2.13	2.83	3.54	4.25	4.96
	1	2.28	2.98	3.69	4.40	5.11	5.82
	2	3.13	3.84	4.55	5.26	5.97	6.68
	3	3.99	4.70	5.41	6.12	6.83	7.53
	4	4.85	5.56	6.27	6.98	7.68	8.39
	5	5.71	6.42	7.13	7.83	8.54	9.25
	6	6.57	7.28	7.98	8.69	9.40	10.1

hypotensive strategy							
P_{aw}		PIP					
		10	**15**	**20**	**25**	**30**	**35**
PEEP	**0**	0.83	1.25	1.67	2.08	2.50	2.92
	1	1.75	2.17	2.58	3.00	3.42	3.83
	2	2.67	3.08	3.50	3.92	4.33	4.75
	3	3.58	4.00	4.42	4.83	5.25	5.67
	4	4.50	4.92	5.33	5.75	6.17	6.58
	5	5.42	5.83	6.25	6.67	7.08	7.50
	6	6.33	6.75	7.17	7.58	8.00	8.42

Barring the most drastic possible scenario (excellent compliance and very low peak pressure) per the general strategy, poor compliance and high peak pressure with transition to the hypotension strategy; paired with keeping PEEP constant), we can see that the hypotension strategy tends to give lower numbers for mean airway pressure. While it is likely that overall compliance will decrease and thus peak inspiratory pressure will increase as we move from left to right (due to higher tidal volume with the hypotension strategy), guesstimating to what degree that will happen seems unfair without actual experimental data. There may also be a mathematical model based on this idea that could identify cases where mean airway pressure isn't actually decreased with the hypotension strategy, but given that this is just one of three reasons to use the strategy (the other two being lower percentage of time at decreased preload and less dead space), it seems OK for now.

At this point there are no experimental data (at least that we are aware of) to show to what extent this type of thing has on cardiac output or other parameters of hemodynamic function, but given the logical

[579] Chiefetz, 2014; Luecke & Pelosi, 2005 - Both articles get into the details of intrathoracic pressure related to positive-pressure ventilation and the many interactions and consequences involved

[580] In the prior version of this manual we never specified which rate we used for the hypotension strategy chart; turns out we did it with a rate of 8/min even though we used a rate of 10/min in the ongoing example in this section; it's all correct now (pretty sure…) and the general trend still holds true

sequence of events that we already outlined it seems like a step in the right direction for the hypotensive patient or one at risk for becoming so.

Just to summarize things for this section: the hypotension strategy includes shorter inspiratory time, increased tidal volume, lower respiratory rate, and keeping PEEP to the lowest level needed to maintain oxygenation. We discussed the idea of percentage of decreased preload (%TaDP) back in the section on hypotension and then we added to that just now the idea that this approach results in both less dead space and a generally lower mean airway pressure. And while PEEP is a major contributor to mean airway pressure, it also serves to maintain oxygenation; this means we ought to use caution in titrating it all the way down to zero.

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